Devi, S.
09/887773

07jun02 10:40:07 User219783 Session D1829.1

SYSTEM:OS - DIALOG OneSearch
File 35:Dissertation Abs Online 1861-2002/May
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File 440:Current Contents Search(R) 1990-2002/Jun 07
(c) 2002 Inst for Sci Info
File 348:EUROPEAN PATENTS 1978-2002/May W04
(c) 2002 European Patent Office
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(c) 2002 Thomson Derwent & ISI
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Derwent announces file enhancements. Please see HELP NEWS 357.
File 113:European R&D Database 1997
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*File 113: This file is closed (no updates)

Set	Items	Description
Set	Items	Description
S3	2985	COXIELLA OR BURNETII OR QFA OR ((QF OR Q(W)FEVER)(10N)ANTI-GEN? ?) OR ANTIGEN?(W)COMPONENT? ?
S9	13	S3 AND (DIABET? OR IDDM OR COXIELLOS? OR (AUTOIMMUN? OR AUTO(W)IMMUN?)(5N)(DISEAS? OR DISORDER? ?))(5N)(TREAT? OR THERAP?)
S10	11	RD (unique items)

-key terms

>>>No matching display code(s) found in file(s): 65, 113

10/3,AB/1 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
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08607581 References: 19
TITLE: Appearance of Graves' disease after percutaneous ethanol injection for the treatment of hyperfunctioning thyroid adenoma
AUTHOR(S): Monzani F (REPRINT); DelGuerra P; Caraccio N; Casolaro A; Lippolis PV; Goletti O
CORPORATE SOURCE: UNIV PISA,MED CLIN 2, IST CLIN MED 2, VIA ROMA 67/I-56126 PISA//ITALY/ (REPRINT); UNIV PISA,DIPARTIMENTO CHIRURG/I-56126 PISA//ITALY/
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF ENDOCRINOLOGICAL INVESTIGATION, 1997, V20, N5 (MAY), P294-298
GENUINE ARTICLE#: XH963
PUBLISHER: EDITRICE KURTIS S R L, VIA LUIGI ZOJA, 30-20153 MILANO, ITALY
ISSN: 0391-4097
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: In this report we describe an unusual patient with hyperfunctioning thyroid adenoma in whom percutaneous ethanol injection (PEI) therapy was followed by typical Graves' disease, His history revealed the presence of a sister with Hashimoto's thyroiditis. Tc99-m thyroid scintiscan showed focal uptake in the nodule, with suppression of extranodular parenchyma. PEI therapy was followed by the development of severe hyperthyroidism. One month after a second PEI cycle, recurrence of hyperthyroidism associated with diffuse Tc99-m uptake by the gland was observed, TSH-receptor and thyroglobulin autoantibodies were undetectable before PEI therapy, appeared during the first cycle, and showed a further increase after the second PEI therapy cycle. Though spontaneous switch to Graves' disease cannot be excluded in patients with toxic nodules, the massive release of thyroid materials from follicular cells, among these TSH-receptor *antigenic"** *components"** partially denatured by ethanol, may indeed trigger an autoimmune response to the TSH-receptor, thus accounting for this observation. Patients with possible autoimmune disposition, as selected by familiar history and/or laboratory markers should be carefully monitored during PEI treatment. (C) 1997, Editrice Kurtis.

10/3,AB/2 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

06421290 References: 17
TITLE: *COXIELLA"** *BURNETII"**, ENDOCARDITIS ON A MECHANICAL VALVE PROSTHESIS - TWO CASE REPORTS
AUTHOR(S): STCHEPINSKY O; PAPO T; AMOYAL P; HUISMAN JP; THEODOSE Y; GAULTIER Y; ALEXANDRE L; PIETTE JC
CORPORATE SOURCE: CTR WILLIAM HARVEY/F-50190 ST MARTIN AUBIGNY//FRANCE/ (Reprint); CHU PITIE SALPETRIERE,SERV MED INTERNE/F-75651 PARIS 13//FRANCE/; HOP MEM FRANCE ETATS UNIS,SERV CARDIOL/F-75651 ST LOUIS//FRANCE/; CABINET MED/F-50190 PERIERS//FRANCE/
PUBLICATION: ARCHIVES DES MALADIES DU COEUR ET DES VAISSEAUX, 1995, V88, N4 (APR), P511-515
GENUINE ARTICLE#: QY396
ISSN: 0003-9683
LANGUAGE: FRENCH DOCUMENT TYPE: NOTE

ABSTRACT: The authors report two cases of prosthetic valve endocarditis due to *Coxiella"** *burnetii"**. The histories were chronic and complex suggesting an *auto"**-*immune"** *disease"** : prolonged reccurent fever despite antibiotic *therapy"** with a biological inflammatory syndrome whilst blood cultures remained negative. The first patient presented with prosthetic valve dehiscence and acute glomerulonephritis. The second patient had coagulation defects with prosthetic valve thrombosis, mesenteric adenopathy and congestive cardiac failure without prosthetic valve dysfunction. In suspected endocarditis with negative blood cultures, serological tests should be extended to intracellular pathogens difficult to identify and justifying specific and prolonged bactericidal therapy (fluoroquinolones, cyclines, rifampincine). Long-term serological surveillance is essential even when the outcome could have led to the termination of antibiotic therapy. Usually, antibiotic therapy provides a bacteriological cure, but treatment has to be continued for at least 3 years, and, in some patients, all their lives. Valve replacement is reserved for haemodynamic complications of the pathology which determine the ultimate prognosis.

10/3,AB/3 (Item 1 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

01238306
Selective immune down regulation (SIDR) mediated transplantation processes
Selektive Immununterdruckungverfahren zur Transplantation
Procedes de regulation immunitaire negative pour la transplantation
PATENT ASSIGNEE:
ENZO THERAPEUTICS, INC., (1726590), 60 Executive Boulevard, Farmingdale, New York 11735-4716, (US), (Applicant designated States: all)
INVENTOR:
Rabbani, Elazar, 69 Fifth Avenue, no. 19A, New York, NY 10003, (US)
Ilan, Yaron, 4/5 Yehoshua Bitzur Street, Givat Masua, 96400 Jerusalem, (IL)
Roy-Chowdhury, Jayanta, 139 Woodhollow Lane, New Rochelle, NY 10804-3435, (US)
Engelhardt, Dean L., 173 Riverside Drive, no. 6D, New York, NY 10024-1616, (US)
LEGAL REPRESENTATIVE:
VOSSIUS & PARTNER (100314), Siebertstrasse 4, 81675 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 1072271 A2 010131 (Basic)
EP 1072271 A3 011004
APPLICATION (CC, No, Date): EP 2000115423 000717;
PRIORITY (CC, No, Date): US 356294 990716
DESIGNATED STATES: AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
EXTENDED DESIGNATED STATES: AL; LT; LV; MK; RO; SI
INTERNATIONAL PATENT CLASS: A61K-039/00; A61K-035/12; A61P-037/06

ABSTRACT EP 1072271 A2
This invention provides for unique immune modulation applications in transplantation processes, as well as in disease intervention and prevention directed to graft-versus-host rejection as well eliminating undesirable immunological effects resulting from immunization and vaccination. Also provided are treatments for other diseases, including Crohn's disease, primary schlerosing cholangitis, primary biliary cirrhosis, atheroschlerosis, etc. This invention also provides unique compositions for carrying out such processes.
ABSTRACT WORD COUNT: 64
NOTE:
Figure number on first page: NONE

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	200105	1537
SPEC A	(English)	200105	15096
Total word count - document A			16633
Total word count - document B			0
Total word count - documents A + B			16633

10/3,AB/4 (Item 2 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

01221350
METHOD FOR JUDGING AUTOIMMUNE DISEASE, METHOD FOR DETECTING ANTI-Reg PROTEIN AUTOANTIBODY AND DIAGNOSTICS FOR AUTOIMMUNE DISEASES
VERFAHREN ZUM BEWERTEN VON AUTOIMMUNKRANKHEITEN, VERFAHREN ZUM NACHWEIS VON GEGEN Reg GERICHTETEN AUTOANTIKORPERN
PROCEDE D'EVALUATION DE MALADIES AUTO-IMMUNES, PROCEDE DE DETECTION D'ANTICORPS DE PROTEINE ANTI-Reg ET DIAGNOSTICS POUR MALADIES AUTO-IMMUNES
PATENT ASSIGNEE:
HITACHI CHEMICAL COMPANY, LTD., (1192832), Shinjuku-Mitsui Building, 1-1, Nishishinjuku 2-chome, P.O. Box 233, Shinjuku-ku, Tokyo 163-0449, (JP), (Applicant designated States: all)
Okamoto, Hiroshi, (3146630), 15-3-205, Tsunogoro 2-chome, Aoba-ku, Sendai-shi, Miyagi 980-0874, (JP), (Applicant designated States: all)
INVENTOR:
OKAMOTO, Hiroshi, 15-3-205, Tsunogoro 2-chome, Aoba-ku, Sendai-shi, Miyagi 980-0874, (JP)
LEGAL REPRESENTATIVE:
Bannerman, David Gardner et al (28001), Withers & Rogers, Goldings House, 2 Hays Lane, London SE1 2HW, (GB)
PATENT (CC, No, Kind, Date): EP 1167974 A1 020102 (Basic)
WO 200062066 001019
APPLICATION (CC, No, Date): EP 2000915389 000406; WO 2000JP2245 000406
PRIORITY (CC, No, Date): JP 9999963 990407
DESIGNATED STATES: AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: G01N-033/564

ABSTRACT EP 1167974 A1
A method for judging an autoimmune disease by detecting the existence of an anti-Reg protein autoantibody in a specimen; and a method for judging insulin-dependent or non-insulin-dependent diabetes mellitus.
A method for detecting an anti-Reg protein autoantibody by bringing into a specimen into contact with an *antigen"** *component"** and detecting the formation of an immune complex.
A reagent for diagnosing autoimmune disease which contain an *antigen"** *component"** capable of binding specifically to the anti-Reg protein autoantibody; and a reagent for diagnosing insulin-dependent or non-insulin-dependent diabetes mellitus.
ABSTRACT WORD COUNT: 87
NOTE:
Figure number on first page: 0001
LANGUAGE (Publication,Procedural,Application): English; English; Japanese
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	200201	669
SPEC A	(English)	200201	7808
Total word count - document A			8477
Total word count - document B			0
Total word count - documents A + B			8477

10/3,AB/5 (Item 3 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00889630

USE OF *COXIELLA"** BACTERIA TO *TREAT"** *AUTOIMMUNE"** *DISEASE"**
Verwendung Von *Coxiella"** Bakterien zur Behandlung von Autoimmunkrankheiten
UTILISATION DE BACTERIES *COXIELLA"** POUR TRAITER DES MALADIES AUTO-IMMUNES
PATENT ASSIGNEE:
THE AUSTRALIAN NATIONAL UNIVERSITY, (209901), , Acton, Australian Capital Territory 2601, (AU), (applicant designated states: AT;BE;CH;DE;DK;ES;FI;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
INVENTOR:
COWDEN, William, Butler, 56 Urambi Village, Kambah, ACT 2902, (AU)
LAFFERTY, Kevin, John, 63 McColloch Street, Curtin, ACT 2605, (AU)
GAZDA, Lawrence, Scott, 19/30 Springvale Drive, Hawker, ACT 2614, (AU)
LEGAL REPRESENTATIVE:
Maschio, Antonio et al (77501), D Young & Co, 21 New Fetter Lane, London EC4A 1DA, (GB)
PATENT (CC, No, Kind, Date): EP 886528 A1 981230 (Basic)
EP 886528 A1 990602
WO 9733614 970918
APPLICATION (CC, No, Date): EP 97906937 970314; WO 97AU161 970314
PRIORITY (CC, No, Date): AU 96PN8703 960314
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
INTERNATIONAL PATENT CLASS: A61K-039/118; A61K-039/02;
NOTE:
No A-document published by EPO
LANGUAGE (Publication,Procedural,Application): English; English; English

10/3,AB/6 (Item 4 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00754357
METHODS AND COMPOSITIONS FOR THE SPECIFIC COAGULATION OF TUMORAL VASCULATURE
VERFAHREN UND ZUSAMMENSETZUNGEN FUR DIE SPEZIFISCHE KOAGULATION VON TUMORGEFASSEN
PROCEDES ET COMPOSITIONS POUR LA COAGULATION SPECIFIQUE DE VAISSEAUX TUMORAUX
PATENT ASSIGNEE:
BOARD OF REGENTS THE UNIVERSITY OF TEXAS SYSTEM, (266340), 201 West 7th Street, Austin, Texas 78701, (US), (Proprietor designated states: all)
The Scripps Research Institute, (1467562), 10550 North Torrey Pines Road, La Jolla, CA 92037, (US), (Proprietor designated states: all)
INVENTOR:
THORPE, Philip E., 6918 Westlake Avenue, Dallas, TX 75214, (US)
EDGINGTON, Thomas S., 2362 Avenida de la Playa, La Jolla, CA 92037, (US)
LEGAL REPRESENTATIVE:
Gowshall, Jonathan Vallance et al (61531), FORRESTER & BOEHMERT Franz-Joseph-Strasse 38, 80801 Munchen, (DE)
PATENT (CC, No, Kind, Date): EP 771216 A1 970507 (Basic)
EP 771216 B1 010117
WO 9601653 960125
APPLICATION (CC, No, Date): EP 95923817 950607; WO 95US7439 950607
PRIORITY (CC, No, Date): US 273567 940711
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE

INTERNATIONAL PATENT CLASS: A61K-047/48
NOTE:
No A-document published by EPO
Figure number on first page: NONE
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	200103	2319
CLAIMS B	(German)	200103	2089
CLAIMS B	(French)	200103	2850
SPEC B	(English)	200103	45787
Total word count - document A			0
Total word count - document B			53045
Total word count - documents A + B			53045

10/3,AB/7 (Item 5 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00742411
Device and process for cell capture and recovery
Vorrichtung und Verfahren zur Fixierung und Ruckgewinnung von Zellen
Appareil et procede de saisie et de recuperation de cellules
PATENT ASSIGNEE:
RORER PHARMACEUTICAL PRODUCTS INC., (2447810), 3711 Kennett Pike, Suite 200, Greenville, Delaware 19807, (US), (Proprietor designated states: all)
INVENTOR:
Okarma, Thomas B., 1651 Portola, Palo Alto, California 94306, (US)
LEGAL REPRESENTATIVE:
Goldin, Douglas Michael et al (31061), J.A. KEMP & CO. 14 South Square Gray's Inn, London WC1R 5JJ, (GB)
PATENT (CC, No, Kind, Date): EP 701130 A2 960313 (Basic)
EP 701130 A3 960320
EP 701130 B1 020522
APPLICATION (CC, No, Date): EP 95118035 900628;
PRIORITY (CC, No, Date): US 374091 890629
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; NL; SE
RELATED PARENT NUMBER(S) - PN (AN):
EP 405972 (EP 90307080)
INTERNATIONAL PATENT CLASS: G01N-033/543; G01N-033/569; G01N-033/577; A61L-002/08; C12M-001/26; C12M-001/12; C12M-001/14

ABSTRACT EP 701130 A2
A method of stabilizing and sterilizing a device for the capture and recovery of cells is provided. The device consists of a biologically active substance immobilized on a polymeric surface.
ABSTRACT WORD COUNT: 39
NOTE:
Figure number on first page: NONE

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS A	(English)	EPAB96	206
CLAIMS B	(English)	200221	215
CLAIMS B	(German)	200221	228

CLAIMS B	(French)	200221	250
SPEC A	(English)	EPAB96	10544
SPEC B	(English)	200221	8004
Total word count - document A			10751
Total word count - document B			8697
Total word count - documents A + B			19448

10/3,AB/8 (Item 6 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00673853
STRESS PROTEINS AND USES THEREFOR
STRESSPROTEINE UND IHRE VERWENDUNG
PROTEINES DU STRESS ET LEURS UTILISATIONS
PATENT ASSIGNEE:
WHITEHEAD INSTITUTE FOR BIOMEDICAL RESEARCH, (782030), Nine Cambridge Center, Cambridge, MA 02142, (US), (Proprietor designated states: all)
INVENTOR:
YOUNG, Richard, A., 5 Sawmill Brook Road, Winchester, MA 01890, (US)
LEGAL REPRESENTATIVE:
Price, Vincent Andrew et al (79513), FRY HEATH & SPENCE The Old College 53 High Street, Horley Surrey RH6 7BN, (GB)
PATENT (CC, No, Kind, Date): EP 700445 A1 960313 (Basic)
EP 700445 B1 020123
WO 9429459 941222
APPLICATION (CC, No, Date): EP 94919384 940606; WO 94US6362 940606
PRIORITY (CC, No, Date): US 73381 930604
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT; SE
RELATED DIVISIONAL NUMBER(S) - PN (AN):
(EP 2001203598)
INTERNATIONAL PATENT CLASS: C12N-015/62; C07K-014/00; A61K-039/295; A61K-039/04
NOTE:
No A-document published by EPO
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	200204	468
CLAIMS B	(German)	200204	434
CLAIMS B	(French)	200204	554
SPEC B	(English)	200204	7900
Total word count - document A			0
Total word count - document B			9356
Total word count - documents A + B			9356

10/3,AB/9 (Item 7 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00431147
Process for cell capture and recovery
Verfahren zur Fixierung und Ruckgewinnung von Zellen
Procede de saisie et de recuperation de cellules
PATENT ASSIGNEE:

RORER PHARMACEUTICAL PRODUCTS INC., (2447810), 3711 Kennett Pike, Suite 200, Greenville, Delaware 19807, (US), (applicant designated states: AT;BE;CH;DE;DK;ES;FR;GB;GR;IT;LI;LU;NL;SE)
INVENTOR:
Okarma, Thomas B., 1651 Portola, Palo Alto, California 94306, (US)
LEGAL REPRESENTATIVE:
Goldin, Douglas Michael et al (31061), J.A. KEMP & CO. 14 South Square Gray's Inn, London WC1R 5LX, (GB)
PATENT (CC, No, Kind, Date): EP 405972 A1 910102 (Basic)
EP 405972 B1 990506
APPLICATION (CC, No, Date): EP 90307080 900628;
PRIORITY (CC, No, Date): US 374091 890629
DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: C12N-005/00; A61K-035/00;

ABSTRACT EP 405972 A1
Devices, processes and compositions are provided for effective separation of cells from a mixture of cells, where depletion or positive selection may be employed to provide a cellular population of interest. Of particular utility is the separation of cells from peripheral blood mononuclear cells, where members of the lymphoid or myeloid lineages may be isolated and used for research, diagnosis or therapy. Also of interest are cellular separation from bone marrow, tumor suspensions or lymphoid tissue suspensions, where cells can be isolated and used for a variety of purposes. The separated cells may be homogeneous, free of exogenous biologicals, viable, capable of replication and exhibit their full complement of biological activities. Multiple phenotypes can be captured simultaneously. Captured cells can be specifically activated with cytokines and antigens to provide cells which are MHC restricted and have antigen-specific effector functions.
ABSTRACT WORD COUNT: 143

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	9918	667
CLAIMS B	(German)	9918	659
CLAIMS B	(French)	9918	786
SPEC B	(English)	9918	10416
Total word count - document A			0
Total word count - document B			12528
Total word count - documents A + B			12528

10/3,AB/10 (Item 8 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00409818
STRESS PROTEINS AND USES THEREFOR.
STRESSPROTEINE UND VERWENDUNGEN DAFUR.
PROTEINES DE STRESS ET LEURS UTILISATIONS.
PATENT ASSIGNEE:
WHITEHEAD INSTITUTE FOR BIOMEDICAL RESEARCH, (782030), Nine Cambridge Center, Cambridge, MA 02142, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)
MEDICAL RESEARCH COUNCIL, (791452), 20 Mount Pleasant, London W1N 4AL, (GB), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)

INVENTOR:
YOUNG, Richard, A., 5 Sawmill Brook Road, Winchester, MA 01890, (US)
YOUNG, Douglas, 44 Lawnclose Ruislip, Middlesex HA4 6ED, (GB)
LEGAL REPRESENTATIVE:
Price, Vincent Andrew et al (79513), FRY HEATH & SPENCE The Old College 53 High Street, Horley Surrey RH6 7BN, (GB)
PATENT (CC, No, Kind, Date): EP 419569 A1 910403 (Basic)
EP 419569 B1 950906
WO 8912455 891228
APPLICATION (CC, No, Date): EP 89907594 890615; WO 89US2619 890615
PRIORITY (CC, No, Date): US 207298 880615
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: A61K-038/00; A61K-039/04;
NOTE:
No A-document published by EPO
LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	EPAB95	248
CLAIMS B	(German)	EPAB95	246
CLAIMS B	(French)	EPAB95	313
SPEC B	(English)	EPAB95	5793
Total word count - document A			0
Total word count - document B			6600
Total word count - documents A + B			6600

10/3,AB/11 (Item 9 from file: 348)
DIALOG(R)File 348:EUROPEAN PATENTS

00218087
Method of conferring immunotolerance to a specific antigen.
Verfahren zur Verleihung von Immuntoleranz gegen ein spezifisches Antigen.
Methode pour conferer une tolerance immunologique vis-a-vis d'un antigene specifique.
PATENT ASSIGNEE:
THE BOARD OF TRUSTEES OF THE LELAND STANFORD JUNIOR UNIVERSITY, (242250), Encina 105 Stanford University, Stanford California 94305, (US), (applicant designated states: AT;BE;CH;DE;FR;GB;IT;LI;LU;NL;SE)
INVENTOR:
Fathman, C. Garrison, 570 Oak Knoll Lane, Menlo Park California 94025, (US)
LEGAL REPRESENTATIVE:
Harrison, David Christopher et al (31531), MEWBURN ELLIS & CO 2/3 Cursitor Street, London EC4A 1BQ, (GB)
PATENT (CC, No, Kind, Date): EP 200412 A2 861105 (Basic)
EP 200412 A3 880330
EP 200412 B1 910612
APPLICATION (CC, No, Date): EP 86302781 860415;
PRIORITY (CC, No, Date): US 724063 850417
DESIGNATED STATES: AT; BE; CH; DE; FR; GB; IT; LI; LU; NL; SE
INTERNATIONAL PATENT CLASS: A61K-045/06; A61K-039/395; A61K-039/00; C07K-017/02; C12N-005/00;

ABSTRACT EP 200412 A2
A method of selectively suppressing the immune system and conferring immunotolerance against a specific antigen by interferring with the L3T4

differentiation antigens on helper T cells is described. Simultaneous administration of a binding moiety specific for the L3T4 equivalent in the subject species and a specific antigen results in a dimished ability of the subject to respond immunologically to the antigen, whether or not the subject has been exposed previously to the antigen.
ABSTRACT WORD COUNT: 75

LANGUAGE (Publication,Procedural,Application): English; English; English
FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	EPBBF1	359
CLAIMS B	(German)	EPBBF1	382
CLAIMS B	(French)	EPBBF1	380
SPEC B	(English)	EPBBF1	6103
Total word count - document A			0
Total word count - document B			7224
Total word count - documents A + B			7224

Set	Items	Description
S3	2985	COXIELLA OR BURNETII OR QFA OR ((QF OR Q(W)FEVER)(10N)ANTIGEN? ?) OR ANTIGEN?(W)COMPONENT? ?
S4	110	S3 AND (DIABET? OR IDDM OR COXIELLOS? OR (AUTOIMMUN? OR AUTO(W)IMMUN?)(5N)(DISEAS? OR DISORDER? ?))
S14	68	S4/TI,DE,MAJ
S15	62	S14 NOT S9
S16	30	RD (unique items)

>>>No matching display code(s) found in file(s): 65, 113

16/3,AB/1 (Item 1 from file: 35)
DIALOG(R)File 35:Dissertation Abs Online

785334 AAD8214194
DETECTION, ISOLATION AND CHARACTERIZATION OF *ANTIGEN"** *COMPONENTS"** OBTAINED FROM IMMUNE COMPLEXES IN HUMAN BREAST CANCER
Author: KOESTLER, THOMAS PAUL
Degree: PH.D.
Year: 1982
Corporate Source/Institution: STATE UNIVERSITY OF NEW YORK AT BUFFALO (0656)
Source: VOLUME 43/03-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 675. 130 PAGES

Rabbits tolerant to human immunoglobulin G were used to raise antisera against the Raji cell-bound circulating immune complexes from human breast cancer sera. After solid-phase adsorption treatment with glutaraldehyde-cross-linked normal human plasma, acetone-extracted normal liver tissue powder, and glutaraldehyde-fixed Raji cells, one antiserum reacted specifically with breast tissue extracts but not with extracts of other tissues, as examined by a counterimmunoelectrophoresis technique. Immunological reactivity of the treated antiserum was removed by incubation with normal, primary, or metastatic breast tumor tissue extracts. Incubation with normal human serum or extracts derived from tissues other than the breast showed no neutralizing effect on the antibodies. This specific antiserum reagent was used in a modification of the Raji cell radioimmunoassay. Raji cells were incubated with sera from cancer patients

or normal controls and then reacted with ('125)I-labeled F(ab')(,2) fraction of the treated antiserum reagent. The amount of ('125)I-F(ab')(,2) bound was then determined. Although all sera exhibited elevated circulating immune complexes by the conventional Raji cell radioimmunoassay, 14 of 18 breast carcinoma sera demonstrated a significant uptake when compared with the normal population group as opposed to five (three lung and two colon) of 29 other cancer sera examined ($p < 0.001$). An immunologically reactive breast tissue-associated antigen, purified from malignant breast tumor or normal breast tissue extracts with the use of antiserum reagent, exhibited an apparent molecular weight of 85,000 by sodium dodecyl sulfate; polyacrylamide gel electrophoresis and a pI value 4.9 (+OR-) 0.2. These results demonstrated that a breast tissue-associated antigen rather than a breast tumor-associated neoantigen, was involved in circulating immune complexes of breast cancer patients as detected by Raji cell immunoassay. It also implied the occurrence of disease-related autoimmunity in human breast cancer.

16/3,AB/2 (Item 1 from file: 77)
DIALOG(R)File 77:Conference Papers Index

Supplier Accession Number: 83053955 V11N1
Non-antigen components of circulating immune complexes (CIC) in sera of type 1 diabetics
Braun, F.J.; Kratzch, G.; Krapf, F.
European Association for the Study of Diabetes, 19th Annual Meeting 8330740 Oslo, Norway 14-17 Sep 83
European Association for the Study of Diabetes (EASD)
Abstracts in "Diabetologia", Aug. 1983, Springer-Verlag, 175 Fifth Ave., New York, NY 10010, USA, Abstract No. 49

16/3,AB/3 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

15374345 PASCAL No.: 02-0062549
Infection of Vero cells with *Coxiella"** *burnetii"** phase II : relative intracellular bacterial load and distribution estimated by confocal laser scanning microscopy and morphometry
ZAMBONI Dario S; MORTARA Renato A; RABINOVITCH Michel
Departamento de Microbiologia, Imunologia e Parasitologia, Escola Paulista de Medicina, UNIFESP. Rua Botucatu, 862, 6 Degree andar, Sao Paulo, SP 04023-062, Brazil
Journal: Journal of microbiological methods, 2001, 43 (3) 223-232
Language: English
Coxiella burnetii, the agent of Q fever in man and of coxiellosis in other species, is an intracellular pathogen not yet grown axenically. Confocal laser fluorescence microscopy and morphometry were used to measure relative C. burnetii phase II loads and their intracellular distribution in aldehyde fixed and DAPI stained Vero cell monolayers. The fluorescence of single horizontal optical sections provided useful information on relative loads of bacteria in cells and vacuoles. The relative density of the bacteria in the vacuoles was inferred from ratios of fluorescence to vacuolar section areas. Relative bacterial loads, bacterial densities and section areas of large vacuoles increased exponentially between days 2 and

4 of the infection of gamma -irradiated host cells, stabilized between days 4 and 6, and decreased thereafter. Estimated minimum doubling times were higher for the overall complement of the intracellular organisms (about 12 h) than for bacteria that were confined to larger vacuoles (about 10 h).

16/3,AB/4 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

15245379 PASCAL No.: 01-0413711
Lack of association between Kawasaki syndrome and infection with Rickettsia conorii, Rickettsia typhi, *Coxiella"** *burnetii"** or Ehrlichia phagocytophila group
KAFETZIS Dimitris A; MALTEZOU Helen C; CONSTANTOPOULOU Ioanna; ANTONAKI Georgia; LIAPI Georgia; MATHIOUDAKIS Ioannis
Second Department of Pediatrics, University of Athens, Athens, Greece; Microbiology Laboratory, P. & A. Kyriakou Children's Hospital, Athens, Greece
Journal: (The) Pediatric infectious disease journal, 2001, 20 (7) 703-706
Language: English
Background. The etiology of Kawasaki syndrome (KS) is unknown. Rickettsiae, intracellular microorganisms that invade the vascular endothelium, might cause KS. Objectives. To investigate whether there is an association between KS and infection with Rickettsia conorii, Rickettsia typhi, Coxiella burnetii or Ehrlichia phagocytophila group. Methods. All children who were diagnosed with KS at the University of Athens Second Department of Pediatrics from December, 1999, through November, 2000, were prospectively studied. Paired serum specimens were obtained from all patients and antibody titers against R. conorii, R. typhi, C. burnetii and E. phagocytophila group were assessed by microimmunofluorescence assay. Results. Eleven children with a median age of 2.5 years were included in the study. A 15-month-old child had a 4-fold rise of antibody titers against C. burnetii, which is indicative of acute Q fever. The patient had a history of recent exposure to possible sources of C. burnetii. The remaining patients tested negative for the presence of antibodies against R. conorii, R. typhi, C. burnetii and E. phagocytophila group. Conclusions. Our study does not provide serologic evidence that KS is the result of infection with R. conorii, R. typhi, C. burnetii or E. phagocytophila group. It is suggested that C. burnetii may cause a KS-like illness in young children.

16/3,AB/5 (Item 3 from file: 144)
DIALOG(R)File 144:Pascal

15128610 PASCAL No.: 01-0291136
Anti-nuclear envelope antibodies : Clinical associations
NESHER Gideon; MARGALIT Ruth; ASHKENAZI Yaacov J
Department of Rheumatology Service, Hebrew University Medical School, Jerusalem, Israel; Departments of Internal Medicine and Hematology, Shaare-Zedek Medical Center, Jerusalem, Israel
Journal: Seminars in arthritis and rheumatism, 2001, 30 (5) 313-320
Language: English

Objectives: Characterization of the clinical associations and clinical implications of antibodies reacting with antigens of the nuclear envelope. Methods: Description of an illustrative case and a MEDLINE search-assisted literature review of relevant cases. Results: With indirect immunofluorescence, autoantibodies directed against various antigens of the nuclear envelope stain the nucleus in a ring-like (rim) pattern. Autoantibodies against 5 antigenic components of the nuclear envelope have been described: anti-gp210, p62, lamina, lamina-associated polypeptides, and lamin B receptor. Antibodies to antigens of the nuclear pore complex, such as gp210 and p62, are highly specific (>95%) for primary biliary cirrhosis and may aid in the serologic diagnosis of this condition, especially in cases in which antimitochondrial antibodies are not detectable. In contrast, antilamin antibodies are not disease-specific but seem to be associated with lupus anticoagulant or anticardiolipin antibodies, antiphospholipid syndrome, thrombocytopenia, autoimmune liver diseases, and arthralgia. High-titered antilamin antibodies help to define a subset of lupus patients with antiphospholipid antibodies who are at a lower risk of developing thrombotic events. In addition, preliminary data suggest that the presence of antilamin antibodies may be helpful in the diagnosis of chronic fatigue syndrome. Conclusions: Each of the antibodies reacting with nuclear membrane antigens has its own spectrum of disease associations. Relevance: Determination of anti-nuclear envelope antibody pattern by indirect immunofluorescence, with subsequent determination of the specific antibody, carries important diagnostic and prognostic implications in various autoimmune conditions.

16/3,AB/6 (Item 4 from file: 144)
DIALOG(R)File 144:Pascal

14667027 PASCAL No.: 00-0340230
Distribution of immunoglobulin G (IgG) and A (IgA) subclasses following Q fever vaccination with soluble phase I *Coxiella"** *burnetii"** extract
CAMACHO M T; OUTSCHOORN I; KOVACOVA E; TELLEZ A
Instituto de Salud Carlos III, CNM, Madrid, Spain; Institute of Virology, Slovak Academy of Sciences, Bratislava, Slovakia
Journal: Vaccine, 2000, 18 (17) 1773-1777
Language: English
High levels of IgG1, IgG3 and IgA2 antibodies have been observed in patients with Q fever following Coxiella burnetii infection. This IgG subclass distribution is more typical of viral and autoimmune diseases than of bacterial infections. It seemed, therefore, of interest to carry out a prospective study of the distribution of immunoglobulin subclasses after vaccination with phase I C. burnetii tricloroacetic soluble extracts to detect possible differences with respect to natural infection. The antibody response found in vaccinees was mainly restricted to the IgG1. IgG2 and IgA1 subclasses. These findings confirm differences in isotype distribution when compared to those of patients with acute or chronic Coxiella infections and opens an area of interest with respect to the role of IgA subclasses.

16/3,AB/7 (Item 5 from file: 144)
DIALOG(R)File 144:Pascal

14448955 PASCAL No.: 00-0107825
Circulating anticentromere CENP-A and CENP-B antibodies in patients with diffuse and limited systemic sclerosis, systemic lupus erythematosus, and rheumatoid arthritis
RUSSO K; HOCH S; DIMA C; VARGA J; TEODORESCU M
Department of Microbiology/Immunology, Section of Rheumatology, College of Medicine, University of Illinois at Chicago; TheraTest Laboratories Inc., Chicago, Illinois, United States; La Jolla Institute for Experimental Medicine, La Jolla, California, United States
Journal: Journal of rheumatology, 2000, 27 (1) 142-148
Language: English
Objective, To determine the disease sensitivity and specificity of testing for autoantibodies against 2 of the 3 main human centromere antigenic components. CENP-A and CENP-B (recombinant, expressed in baculovirus). Methods. ELISA with CENP-A and CENP-B antigens were used to test 45 sera showing a centromere pattern by immunofluorescence (IFA) and sera from 96 patients with systemic sclerosis (SSc), subdivided into diffuse (dSSc) and limited (ISSc) forms. For controls, the same tests were performed on sera from 100 patients with rheumatoid arthritis (RA). 100 with systemic lupus erythematosus (SLE), and 50 random blood donors. Sera from all the patients with SSc were also tested for the presence of anti-Scl70 antibody by ELISA (bovine antigen and for pattern and titer by IFA (HEp-2 cells). Results. Of the 45 IFA positive sera, 93% were positive for anti-CENP-A and 91% for anti-CENP-B. There was a very good quantitative correlation between the antibody levels against these 2 centromere components ($r = 0.597$; $p < 0.001$). Anti-CENP-A and B were found in 48% of patients with ISSc, and in 11% and 9%, respectively, of those with dSSc. The difference in the frequency of anti-CENP-A between the 2 patient groups was significant (chi-squared, $p < 0.001$). Similar levels of anticentromere staining pattern by IFA were observed for these 2 groups. Anti-Scl70 was elevated in 8% of ISSc and 25% of dSSc patients; this difference was also significant (chi-squared, $p = 0.02$). Neither CENP-A nor CENP-B reacted with IgG from SSc patients containing anti-Scl70. The frequency of abnormal levels in patients with SLE and RA was, respectively, 11% and 3% for anti-CENP-A and 4% and 3% for anti-CENP-B. The reaction of IgG from SLE and RA patients with CENP-A was not inhibited by histone H3, i.e., it was not due to recognition of the histone-like domain in CENP-A. Thus, when 96 SSc patients were compared to 200 patients with RA and SLE, the disease specificity of anti-CENP-A and B was 93% and 96.5%, respectively. Conclusion. In addition to IFA, ELISA tests for CENP-A and CENP-B yield results with similar sensitivity and specificity for the diagnosis of SSc. CENP-A and CENP-B are primarily associated with ISSc. In SSc the autoantibody response is directed simultaneously and with similar amplitude against these 2 components of the centromere structure, whereas in other autoimmune diseases the response is directed mainly against one of the 2 components.

16/3,AB/8 (Item 6 from file: 144)
DIALOG(R)File 144:Pascal

13719450 PASCAL No.: 98-0410734
Nuclear antigen histone H1 is primarily involved in lupus erythematosus

cell formation
SCHETT G; STEINER G; SMOLEN J S
University of Vienna, Vienna, Austria; Ludwig Boltzmann Institute of Rheumatology, Vienna, Austria
Journal: Arthritis and rheumatism, 1998, 41 (8) 1446-1455
Language: English
Objective. To elucidate the nature of the antigen reactive with the "lupus erythematosus (LE) cell factor," the autoantibody involved in the LE cell phenomenon. Methods. Serum samples from systemic lupus erythematosus (SLE) patients who were positive for the LE cell phenomenon (LEc+) and SLE patients who were negative for the LE cell phenomenon (LEc-) were used to characterize the nuclear antigen bound by the LE cell factor, by immunoblotting and immunoprecipitation techniques. Results. All LEc+ sera, but none of the LEc-sera, uniformly reacted with a double band of MW similar 30 kd in nuclear extracts. Depletion of nuclear protein extracts of antigens bound by pooled LEc- serum allowed precipitation of a low molecular weight protein by pooled LEc+ serum. This protein was able to block LE cell formation by LEc+ serum. Based on its reactivity with antihistone antibody and an electrophoretic mobility identical with that of precipitated and purified histone H1, this protein was identified as histone H1. Moreover, all LEc+ sera, but none of the LEc- sera, reacted with purified histone H1 by immunoblotting, whereas other histones were reactive with both types of sera. In addition, purified histone H1, but none of the other histones, strongly inhibited the induction of LE cells by LEc+ serum. Conclusion. Histone H1 represents the major antigenic component recognized by the LE cell factor. Thus, the LE cell phenomenon appears to be due primarily to anti-histone H1 reactivity.

16/3,AB/9 (Item 7 from file: 144)
DIALOG(R)File 144:Pascal

13473456 PASCAL No.: 98-0170490
An explosive outbreak of Q-fever in Jedl'ove Kostol'any, Slovakia
VARGA V
State Health Institute, Nitra, Slovakia
Journal: Central european journal of public health, 1997, 5 (4) 180-182
Language: English
An explosive epidemic of Q-fever that occurred at Jedfove Kostotany (Nitra District) in April 1993, had an unusual mode of transmission, unprecedented in Slovakia. The submitted case-reports can be very instructive for both health workers and the lay public. The bulk of infection was spread in the local pub through contaminated garments of animal attendants assisting abortions and births of goats in a large capacity breeding centre of Gemersan Co. By their repeated visits to the local pub the infection most probably spread to other guests by aerosol. A total of 113 persons (103 males, 10 females) contracted Q-fever. Out of them 95 were infected by contact with the goat attendants (84 %), and 18 were occupational diseases after direct contact with the infected goats. The human epidemic of Q-fever facilitated to trace an ongoing epizootic of coxiellosis in the herd of goats of Gemersan breed. In order to stop further spread of infection among humans and to contain the outbreak of coxiellosis among animals extensive epidemic and epizootic measures were taken.

16/3,AB/10 (Item 8 from file: 144)
DIALOG(R)File 144:Pascal

13144634 PASCAL No.: 97-0404454
Appearance of Graves' disease after percutaneous ethanol injection for the treatment of hyperfunctioning thyroid adenoma
MONZANI F; DEL GUERRA P; CARACCIO N; CASOLARO A; LIPPOLIS P V; GOLETTI O
Istituto di Clinica Medica, University of Pisa, Via Roma, Pisa, Italy; Dipartimento di Chirurgia, University of Pisa, Via Roma, Pisa, Italy
Journal: Journal of endocrinological investigation, 1997, 20 (5) 294-298
Language: English
In this report we describe an unusual patient with hyperfunctioning thyroid adenoma in whom percutaneous ethanol injection (PEI) therapy was followed by typical Graves' disease. His history revealed the presence of a sister with Hashimoto's thyroiditis. SUP 9 SUP 9 SUP - SUP m Tc thyroid scintiscan showed focal uptake in the nodule, with suppression of extranodular parenchyma. PEI therapy was followed by the development of severe hyperthyroidism. One month after a second PEI cycle, recurrence of hyperthyroidism associated with diffuse SUP 9 SUP 9 SUP - SUP m Tc uptake by the gland was observed. TSH-receptor and thyroglobulin autoantibodies were undetectable before PEI therapy, appeared during the first cycle, and showed a further increase after the second PEI therapy cycle. Though spontaneous switch to Graves' disease cannot be excluded in patients with toxic nodules, the massive release of thyroid materials from follicular cells, among these TSH-receptor antigenic components partially denatured by ethanol, may indeed trigger an autoimmune response to the TSH-receptor, thus accounting for this observation. Patients with possible autoimmune disposition, as selected by familiar history and/or laboratory markers should be carefully monitored during PEI treatment.

16/3,AB/11 (Item 9 from file: 144)
DIALOG(R)File 144:Pascal

12919807 PASCAL No.: 97-0188329
Alcohol dehydrogenase : A target of humoral *autoimmune"** response in liver *disease"**
MA Y; GAEKEN J; MCFARLANE B M; FOSS Y; FARZANEH F; MCFARLANE I G; MIELI-VERGANI G; VERGANI D
Institute of Hepatology, University College London Medical School, London , United Kingdom; Department of Immunology, King's College School of Medicine and Dentistry, London, England, United Kingdom; Department of Molecular Medicine, King's College School of Medicine and Dentistry, London, England, United Kingdom; Department of Molecular Medicine and Institute of Liver Studies, King's College School of Medicine and Dentistry , London, England, United Kingdom; Department of Child Health, King's College School of Medicine and Dentistry, London, England, United Kingdom
Journal: Gastroenterology : (New York, NY. 1943), 1997, 112 (2) 483-492
Language: English
Background & Aims: Liver-specific membrane lipoprotein (LSP) is a heterogeneous liver preparation that has been widely used to study

autoreactivity in liver disease. The aim of this study was to identify autoantigens in LSP. Methods: Guinea pig anti-LSP serum was used to screen a human liver complementary DNA (cDNA) library. Humoral immune responses to isolated potential autoantigens were investigated by immunoblotting in 91 pediatric patients with various liver diseases, 20 adult patients with alcoholic liver disease and 20 with autoimmune thyroid disease, 37 healthy children, and 20 healthy adults. Results: A 1.6-kilobase cDNA insert isolated from the cDNA library was found to encode amino acids 61-374 of the human alcohol dehydrogenase (ADH)- gamma SUB 1 subunit. Antibodies to this or other ADH subunits were found significantly more frequently in autoimmune liver diseases (19 of 39 patients; 49%), Wilson's disease (5 of 13 patients; 38%), and alcoholic liver disease (10 of 20 patients; 50%) than in normal controls ($P < 0.0001$, $P < 0.005$, and $P < 0.05$, respectively) and correlated with disease activity in autoimmune liver disease. Conclusions: ADH has been identified as a new antigenic component of the LSP using a xenogeneic antiserum to immunoprobe a human cDNA liver library and seems to be a target autoantigen in liver disease. This approach may be useful in identifying other potential autoantigens.

16/3,AB/12 (Item 10 from file: 144)
DIALOG(R)File 144:Pascal

12887942 PASCAL No.: 97-0151250
Cross-reactivity of human IgG anti-F(ab') SUB 2 antibody with DNA and other nuclear antigens
WILLIAMS R C JR; MALONE C C; CIMBALNIK K; PRESLEY M A; ROUX K H; STRELETS L; SILVESTRIS F
University of Florida School of Medicine, Gainesville, France; Florida State University, Tallahassee, United States; University of Bari Medical School, Bari, Italy
Journal: Arthritis and rheumatism, 1997, 40 (1) 109-123
Language: English
Objective. To characterize immunologic specificity and possible antiidiotype activity of IgG anti-F(ab') SUB 2 in normal subjects as well as in patients with active and inactive systemic lupus erythematosus (SLE). Methods. IgG anti-F(ab') SUB 2 and anti-doublestranded DNA (anti-dsDNA) were affinity isolated from immunoadsorption columns of F(ab') SUB 2 and dsDNA linked to Sepharose 4B. Affinity-purified IgG anti-F(ab') SUB 2 (APAF) and affinity-isolated IgG anti-dsDNA (APAD) were tested by enzyme-linked immunosorbent assay (ELISA) for other cross-reacting specificities including anti-Sm, anti-Sm/RNP, and anti- Crithidia binding. Anti-DNA specificity of APAF and APAD was assayed by S1 nuclease treatment of heat-denatured DNA. Rabbit antiidiotypic antisera were prepared by immunization with APAF and APAD from normal subjects and SLE patients and absorption with insolubilized human Cohn fraction II (Fr II). V SUB L and V SUB H regions of 5 monoclonal IgM antibodies with anti-F(ab') SUB 2 /anti-DNA specificity generated by Epstein-Barr virus B cell stimulation were sequenced by polymerase chain reaction and characterized for V SUB H and V SUB L subgroup. APAF and APAD were also examined by high-resolution electron microscopy for possible ring forms indicative of antiidiotypic V-region interactions. Results. APAF from normal subjects, representing 0.08-0.18% of serum IgG, showed striking relative concentrations of both anti-F(ab') SUB 2 and anti-DNA, as well as anti-Sm and anti-Sm/RNP ELISA reactivity. Both APAF and APAD reacting with F(ab') SUB 2 or dsDNA on the

ELISA plate could be cross-inhibited by F(ab') SUB 2 or DNA in solution. Anti-DNA reactivity in normal APAF and APAD was much more sensitive to S1 nuclease treatment than similar fractions from SLE patients. Neither APAF nor APAD from controls produced positive antinuclear immunofluorescence or positive Crithidia staining, whereas these were strongly positive using SLE APAF and APAD. Absorbed rabbit antisera against normal or SLE APAF and APAD showed strong ELISA reactivity against both APAF and APAD, but no residual reactivity with normal Fr II. V SUB L and V SUB H sequencing of monoclonal human IgM antibodies showing both anti-F(ab') SUB 2 and anti-DNA reactivity showed relative V SUB H 3, V kappa 1 or V SUB H 1, V kappa 3 restriction. No evidence of ring forms or V-region "kissing" dimers was obtained when normal or SLE APAD or APAF was examined by high-resolution electron microscopy. Conclusion. IgG anti-F(ab') SUB 2 in both normal subjects and SLE patients represents a polyreactive Ig subfraction with concomitant anti-DNA, anti-Sm, and anti-Sm/RNP specificities. Anti-DNA reactivity in SLE is qualitatively different from that in normal APAD and APAF since normal APAD and APAF anti-DNA is much more sensitive to S1 nuclease digestion of denatured dsDNA. APAF and APAD share distinct V-region antigens which may be related to prominent V SUB H 3 or V SUB H 1 antigenic components. No evidence for in vivo complexing of anti-DNA and anti-F(ab') SUB 2 as ring forms or antiidiotype-IgG complexes was observed during ultrastructural studies. In both normal individuals and SLE patients, APAF may represent a small polyreactive IgG subfraction which also contains antinuclear and anti-DNA specificities.

16/3,AB/13 (Item 11 from file: 144)
DIALOG(R)File 144:Pascal

11955159 PASCAL No.: 95-0134993
Analysis of the Mi-2 autoantigen of dermatomyositis
NILASENA D S; TRIEU E P; TARGOFF I N
Veterans affairs medical cent., Salt Lake City UT, USA
Journal: Arthritis and rheumatism, 1995, 38 (1) 123-128
Language: English
Objective. To determine the biochemical structure and antigenic components of Mi-2 autoantigen, the target of autoantibodies in 15-20% of dermatomyositis patients. Methods. Immunoprecipitation from SUP 3 SUP 5 S-labeled HeLa cell extract, immunoblotting, and purification from bovine thymus by immunoaffinity chromatography. Results. All 46 sera that had anti-Mi-2 autoantibodies demonstrated by immunodiffusion immunoprecipitated a major protein of similar 240 kd. Additional proteins of 200, 150, 72, 65, 63, 50, and 34 kd appeared to be part of the antigen. Fractions of purified bovine Mi-2 with antigenic activity showed high molecular weight bands comparable with immunoprecipitated HeLa Mi-2. Twenty-four of 47 anti-Mi-2 positive sera reacted with the 240-kd protein by immunoblot against anti-Mi-2 immunoprecipitates. Conclusion. Mi-2 antigen consists of multiple proteins, of which the 240-kd protein appears to be the major reactive component

16/3,AB/14 (Item 12 from file: 144)
DIALOG(R)File 144:Pascal

11846156 PASCAL No.: 95-0008617
Analysis of the specificity of anti-PM-Scl autoantibodies
QUN GE; YAJUAN WU; TRIEU E P; TARGOFF I N
Oklahoma medical res. foundation, Oklahoma City, Japan
Journal: Arthritis and rheumatism, 1994, 37 (10) 1445-1452
Language: English
Objective. To compare the specificity of anti-PM-Scl autoantitiodies in serum samples from 43 patients with myositis, scleroderma, or both. Methods. Anti-PM-Scl immunoprecipitates from HeLa cell extract were used as antigen for immunoblot analyses to determine the antigenic components. A series of complementary DNA fragments was expressed in Escherichia coli for immunoblot examination of the reaction with tHe 100-kd protein. Results. The immunoblot against immunoprecipitates was sensitive and specific for detecting reactions with components of the PM-Scl antigen: 42 of 43 sera (97.7%) reacted with the 100-kd, 27 of 43 (62.8%) with the 70-kd, and 5 of 43 (11.6%) with the 37-kd protein (not previously recognized as antigenic)

16/3,AB/15 (Item 13 from file: 144)
DIALOG(R)File 144:Pascal

11242363 PASCAL No.: 94-0060412
Q fever is absent from New Zealand
HILBINK F; PENROSE M; KOVACOVA E; KAZAR J
Ministry agriculture fisheries, cent. animal health lab., Upper Hutt, New Zealand
Journal: International journal of epidemiology, 1993, 22 (5) 945-949
Language: English
To investigate the presence of Coxiella burnetii in sheep and cattle, the two major ruminant populations of New Zealand, its seroprevalence was determined in aborting cattle and sheepdogs. These groups of animals were chosen because of their accessibility and the fact that they would be good indicators for the presence of the organism. A total of 2181 bovine and 12 556 canine samples were all seronegative. On the basis of these results and previous reports it is argued that New Zealand is free of coxiellosis or Q fever

16/3,AB/16 (Item 14 from file: 144)
DIALOG(R)File 144:Pascal

10680289 PASCAL No.: 93-0189586
Cloning and characterization of cDNA coding for a polymyositis-scleroderma overlap syndrome-related nucleolar 100-kD protein
BLUETHNER M; BAUTZ F A
Univ. Heidelberg, inst. molecular genetics, W-6900 Heidelberg, Federal Republic of Germany
Journal: (The) Journal of experimental medicine, 1992, 176 (4) 973-980
Language: English
About 50% of patients with the polymyositis-scleroderma overlap syndrome are reported to have autoantibodies to a nucleolar particle termed PM/Scl. The particle consists of several polypeptides of which two proteins of 75 and 100 kD have been identified as the major antigenic components. Here we report on the cDNA cloning and partial epitope mapping of the 100-kD autoantigen from human placenta and HeLa lambda gt11 libraries. The deduced amino acid sequence encodes a protein of 885 amino acid residues with a

molecular mass of 100. 8 kD

16/3,AB/17 (Item 15 from file: 144)
DIALOG(R)File 144:Pascal

09675625 PASCAL No.: 91-0472753
Glycolipids and complete Freund's adjuvant cause insulitis in rats
BORNSTEIN S R; BOECKMANN M; YASSIN N; SCHERBAUM W A; PFEIFFER E F
Univ. Ulm, Medizinische Klin. Poliklin., Abt. Innere Medizin I, Ulm 7900, Federal Republic of Germany
Journal: Hormone and metabolic research, 1991, 23 (3) 139-140
Language: English
Gangliosides are considered to be the antigenic component reacting with islet cell antibodies which are one of the serological hallmarks of type 1 diabetes mellitus. We therefore imminized rats with glycolipids and Freund's adjuvant and we then studied the histology of the pancreas and the metabolic status in parallel with the development of insulitis

16/3,AB/18 (Item 16 from file: 144)
DIALOG(R)File 144:Pascal

08946808 PASCAL No.: 90-0114945
Incidence and reactivity patterns of skeletal and heart (SH) reactive autoantibodies in the sera of patients with myasthenia gravis
CONNOR R I; LEFVERT A K; BENES S C; LANG R W
Ohio state univ., dep. medical microbiology immunology, Columbus OH 03756, USA
Journal: Journal of neuroimmunology, 1990, 26 (2) 147-157
Language: English
This study describes a new procedure for the extraction of human cardiac muscle which allows direct determination of SH antibody reactivity. Serologic evaluation of 17 patients with MG revealed 9/17 (53%) were seropositive for SH antibody to cardiac muscle. Absorption of selected MG serum samples with cardiac muscle extracts, reduced or eliminated reactivity to skeletal muscle in all cases, confirming the presence of cross-reactive antibodies. Immunoblot analysis of cardiac muscle extracts demonstrated several distinct antigenic components, which were unrelated to the acetylcholine receptor or to previously identified striational muscle proteins

16/3,AB/19 (Item 17 from file: 144)
DIALOG(R)File 144:Pascal

03001520 PASCAL No.: 81-0034980
*COXIELLOSIS"** IN FOULS OF KARNATAKA STATE
STEPHEN S; CHANDRASHEKARA I; RAO K N A
KASTURBA MED. COLL./MANIPAL 576119,INDIA
Journal: INDIAN J. MED. RES., 1980-03, 71 363-364
Language: ENGLISH
ETUDE DU ROLE DES VOLAILLES DANS L'EPIDEMIOLOGIE DE LA FIEVRE Q

16/3,AB/20 (Item 18 from file: 144)
DIALOG(R)File 144:Pascal

02784872 PASCAL No.: 80-0326873
*COXIELLOSIS"** IN REPTILES OF SOUTH KANARA DISTRICT, KARNATAKA
STEPHEN S; ACHYUTHA RAO K N
KASTURBA MED. COLL.,MANIPAL 576119,INDIA
Journal: INDIAN J. MED. RES., 1979-12, 70 937-941
Language: ENGLISH

16/3,AB/21 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

11352181 References: 41
TITLE: Seroprevalence of *coxiellosis"** in cattle, sheep and people in the east of Turkey
AUTHOR(S): Cetinkaya B (REPRINT); Kalender H; Ertas HB; Muz A; Arslan N; Ongor H; Gurcay M
CORPORATE SOURCE: Univ Firat, Fac Vet, /TR-23119 Elazig//Turkey/ (REPRINT); Univ Firat, Fac Vet, /TR-23119 Elazig//Turkey/; Vet Control & Res Inst, /Elazig//Turkey/
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ISSN: 0042-4900
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Serum samples collected randomly from 416 cattle in 48 herds, and 411 sheep in 47 flocks, in eight different locations in the east of Turkey between lune and December 1998, were examined by indirect fluorescent antibody test (IFAT) to determine the prevalence of Q fever. The age, sex, breed, tick control and abortion history of the animals were also recorded. In addition, 102 serum samples were collected from apparently healthy people who were at risk of contracting the disease, such as farmers, veterinarians, abattoir and laboratory workers, and veterinary students. Seropositivity was observed in 5.8 per cent (24/416) of the cattle in 17 (35.4 per cent) of the herds and in 10.5 per cent (43/411) of the sheep in 21 (44.7 per cent) of the flocks. Eight of the 102 people were seropositive, with the highest prevalence (12.0 per cent) in farmers and abattoir workers. All the seropositive farmers had seropositive animals. None of the laboratory workers or veterinary students was seropositive.

16/3,AB/22 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

10044001 References: 19
TITLE: Occurrence of bovine *coxiellosis"** in the district of Bardejov, Eastern Slovakia
AUTHOR(S): Rehacek J (REPRINT); Kocianova E; Kovacova E; Kapitancik B; Jurcina A; Nad O; Licko P
CORPORATE SOURCE: SLOVAK ACAD SCI,VIROL USTAV, DUBRAVSKA CESTA 9/BRATISLAVA

84246//SLOVAKIA/ (REPRINT); UNIV VET MED,/KOSICE//SLOVAKIA/; DEPT STATE VET CARE,DIST OFF/BARDEJOV//SLOVAKIA/
PUBLICATION TYPE: JOURNAL
PUBLICATION: VETERINARNI MEDICINA, 1998, V43, N11 (AUG), P325-330
GENUINE ARTICLE#: 142UX
PUBLISHER: INST AGRICULTURAL FOOD INFORMATION, SLEZSKA 7, PRAGUE 120 56, CZECH REPUBLIC
ISSN: 0375-8427
LANGUAGE: Czech DOCUMENT TYPE: ARTICLE

ABSTRACT: In the years 1996 to 1997 the occurrence of coxiellosis was studied in the district of Bardejov. The case of abortion and two cases of retention of secundines in dairy cows in the Agricultural Go-operative (RD) Zborov in April 1996 and agglutination antibodies against Coxiella burnetii in 33% of the herd (328 cows) of which these animals originated, served as a basis for mass inoculation of cattle by the vaccine Bodibion a.u.v. in May 1996. Cases of Q fever in humans were not recorded at all. Neither antibodies against C. burnetii were found in nine tested employees of the central farm Zborov. There were no clinical manifestations of coxiellosis in studied animals, the level of antibodies against C. burnetii in 10 long-time studied animals which had high levels of antibodies in the first sampling was gradually falling and even antibodies disappeared in some animals towards the end of studied period after Is months (October 1997). The presence of coxiella in milk in three of eight tested dairy cows observed before inoculation and five months after it was not confirmed in further examinations. The causative agent was not found in small mammals (369 tested), neither in ectoparasites (82 tested ticks, 663 mites, 57 fleas and 73 lice). The possible source of infection of cattle in the studied rearing is discussed in the contribution.

16/3,AB/23 (Item 3 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09732409 References: 24
TITLE: Prevalence of *Coxiella"** *burnetii"** infection in dairy cattle with reproductive disorders- Note
AUTHOR(S): To H; Htwe KK; Kako N; Kim HJ; Yamaguchi T; Fukushi H; Hirai K (REPRINT)
CORPORATE SOURCE: GIFU UNIV,DEPT VET MICROBIOL, FAC AGR, 1-1 YANAGIDO/GIFU 5011193//JAPAN/ (REPRINT); GIFU UNIV,DEPT VET MICROBIOL, FAC AGR/GIFU 5011193//JAPAN/
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF VETERINARY MEDICAL SCIENCE, 1998, V60, N7 (JUL), P 859-861
GENUINE ARTICLE#: 108FB
PUBLISHER: JAPAN SOC VET SCI, UNIV TOKYO, 1-1-1 YAYOI, BUNKYO-KU, TOKYO 103, JAPAN
ISSN: 0916-7250
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: The prevalence of Coxiella burnetii infection in 207 cattle with reproductive disorders was studied by using an indirect immunofluorescence (IF) test, nested polymerase chain reaction (PCR) and isolation. IF antibodies to phase I and phase II antigens of C. burnetii were found in 122 (58.9%) and 125 (60.4%) of the sera, respectively, and PCR-positives were found in 8 (3.9%) of the sera and in 51 (24.6%) of the milk samples.

In addition, C, burnetii was isolated from 51 (24.6%) of the milk samples by inoculating laboratory mice. The results indicate that the IF test plus PCR are useful in the diagnosis of bovine coxiellosis. It is difficult to deny that dairy cattle with reproductive disorders would be one of the important reservoirs of C. burnetii responsible for infection in both animal and human populations in Japan.

16/3,AB/24 (Item 4 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09732395 References: 81
TITLE: Advances in the understanding of *Coxiella"** *burnetii"** infection in Japan
AUTHOR(S): Hirai K (REPRINT); To H
CORPORATE SOURCE: GIFU UNIV,DEPT VET MICROBIOL, FAC AGR, 1-1 YANAGIDO/GIFU 5011193//JAPAN/ (REPRINT)
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF VETERINARY MEDICAL SCIENCE, 1998, V60, N7 (JUL), P 781-790
GENUINE ARTICLE#: 108FB
PUBLISHER: JAPAN SOC VET SCI, UNIV TOKYO, 1-1-1 YAYOI, BUNKYO-KU, TOKYO 103, JAPAN
ISSN: 0916-7250
LANGUAGE: English DOCUMENT TYPE: REVIEW

ABSTRACT: Q fever is a zoonotic disease caused by a rickettsia Coxiella burnetii. Since its first description in 1937, the disease has been found to be present in most countries of the world. Serological evidences of Q fever in humans and coxiellosis in animals were reported in Japan in the 1950s, however, systematic studies of the disease did not begin until the report of isolation of C. burnetii from an acute Q fever patient in 1989. In addition to the extensive information about epidemiology of the disease, the understanding of Japanese isolates of C. burnetii is increasing rapidly in recent years. In this review, the epidemiology of the disease along with some characteristics of isolates of C. burnetii in Japan is summarized in five sections, i.e., coxiellosis, Q fever, modes of spread of the infection, laboratory diagnosis of the infection and some characteristics of Japanese isolates. This review includes some recent, unpublished data from our and other groups.

16/3,AB/25 (Item 5 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09409551 References: 28
TITLE: *Coxiellosis"** in domestic and wild birds from Japan
AUTHOR(S): To H (REPRINT); Sakai R; Shirota K; Kano C; Abe S; Sugimoto T; Takehara K; Morita C; Takashima I; Maruyama T; Yamaguchi T; Fukushi H; Hirai K
CORPORATE SOURCE: GIFU UNIV,FAC AGR, DEPT MICROBIOL/GIFU 50111//JAPAN/ (REPRINT); KITASATO UNIV,SCH VET MED & ANIM SCI, DEPT POULTRY DIS/TOWADA/AOMORI 034/JAPAN/; RAKUNO GAKUEN UNIV,SCH VET MED, DEPT VET PUBL HLTH/EBETSU/HOKKAIDO 069/JAPAN/; HOKKAIDO UNIV,SCH VET MED, DEPT VET PUBL HLTH/SAPPORO/HOKKAIDO 060/JAPAN/; AZABU UNIV,FAC ENVIRONM HLTH SCI, DEPT FOOD HYG/KANAGAWA 229//JAPAN/

PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF WILDLIFE DISEASES, 1998, V34, N2 (APR), P310-316
GENUINE ARTICLE#: ZJ559
PUBLISHER: WILDLIFE DISEASE ASSN, INC, 810 EAST 10TH ST, LAWRENCE, KS 66044-8897
ISSN: 0090-3558
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Serological evidence of infection with Coxiella burnetii was found 41 (2%) of 1,951 domestic birds and in 167 (19%) of 863 wild birds from 17 and 5 prefectures in Japan, respectively, by microagglutination (MA) test. The bacteriological evidence of the infection was found in 17 (41%) of 41 domestic birds and 37 (22%) of 167 wild birds by the nested polymerase chain reaction (PCR). In addition, C. burnetii was isolated from five each of serum, spleen and fecal specimens from five jungle crows (Corvus macrorhynchos) (whose sera were positive by both the MA test and PCR) by inoculating laboratory mice. Domestic quail (Coturnix coturnix japonica) (3%), domestic muscovy ducks (Cairina moschata) (3%), domestic chickens (2%), domestic mallards (Anas platyrhynchos domesticus) (2%), carrion crows (Corvus corone) (37%), jungle crows (35%), and wild rock doves (Columba livia) (6%) showed serologic evidence of experience with C. burnetii. There was a tendency for a high prevalence among birds living and/or feeding in close proximity to infected livestock. This suggests that these birds are one of the less important links in maintaining the whole cycle of C. burnetii infection.

16/3,AB/26 (Item 6 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

06319558 References: 13
TITLE: NECROTIZING BRONCHITIS, ANGIITIS, AND AMYLOIDOSIS ASSOCIATED WITH CHRONIC Q FEVER
AUTHOR(S): KAYSER K; WIEBEL M; SCHULZ V; GABIUS HJ
CORPORATE SOURCE: THORAXKLIN,DEPT PATHOL,AMALIENSTR 5/D-69126 HEIDELBERG//GERMANY/ (Reprint); THORAXKLIN,DEPT PNEUMOL/HEIDELBERG//GERMANY/; UNIV MUNICH,INST PHYSIOL CHEM/W-8000 MUNICH//GERMANY/
PUBLICATION: RESPIRATION, 1995, V62, N2 (MAR-APR), P114-116
GENUINE ARTICLE#: QR917
ISSN: 0025-7931
LANGUAGE: ENGLISH DOCUMENT TYPE: NOTE

ABSTRACT: The authors report the clinical, radiological and histological findings in a 63-year-old male patient who developed severe necrotizing bronchitis, necrotizing angiitis, and secondary amyloidosis of the right upper love and intermediate bronchus. The patient expired due to respiratory insufficiency. At the age of 27 years, the patient had had radiotherapy of the mediastinum because of suspected Hodgkin's disease. Acute pneumonia suggestive of Q-fever infection was diagnosed at the age of 48. Progressive restrictive lung disease developed during the last decade. Serological evaluation revealed IgM and IgA high titers against Coxiella burnetii. IgA, complement and amyloid deposits were detected in the walls of small arteries. Bronchial lavage and pleural effusions displayed numerous activated T lymphocytes. Analysis of endogenous lectins revealed alterations of the pulmonary defense system. The clinical history, histological and immunological findings suggest that chronic Q fever may

induce remarkable changes in the immune system, comparable to autoimmune-reactive diseases.

16/3,AB/27 (Item 7 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

05002416 References: 13
TITLE: A SEROLOGIC SURVEY FOR SOME BACTERIAL AND VIRAL ZOONOSES IN GAME ANIMALS IN THE CZECH-REPUBLIC
AUTHOR(S): HUBALEK Z; JURICOVA Z; SVOBODOVA S; HALOUZKA J
CORPORATE SOURCE: ACAD SCI BRNO,INST SYSTEMAT & ECOL BIOL,KVETNA 8/CS-60365 BRNO//CZECHOSLOVAKIA/ (Reprint)
PUBLICATION: JOURNAL OF WILDLIFE DISEASES, 1993, V29, N4 (OCT), P604-607
GENUINE ARTICLE#: MF941
ISSN: 0090-3558
LANGUAGE: ENGLISH DOCUMENT TYPE: NOTE

ABSTRACT: Between 1986 and 1991, sera were collected from 33 roe deer (Capreolus capreolus), 24 red deer (Cervus elaphus), four fallow deer (Dama dama), two mouflon (Ovis musimon), 34 wild boars (Sus scrofa), and 48 hares (Lepus europaeus) shot in two areas of the Czech Republic. Collectively, the sera contained antibodies to Coxiella burnetii (prevalence of 12%), Francisella tularensis (4%), Brucella spp. (2%), Central European tick-borne encephalitis virus (8%), Tahyna (California serogroup) virus (36%), and Calovo (=Batai) virus (23%). We propose that these mammals may play a role in maintaining natural foci of Q-fever, Tahyna fever and Calovo virus infection.

16/3,AB/28 (Item 8 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

03877566 References: 15
TITLE: SEROPREVALENCE OF XOCIELLA-*BURNETII"** AND CHLAMYDIA-PSITTACI IN DOGS
AUTHOR(S): KOCIANOVA E; LISAK V; KOPCOK M
CORPORATE SOURCE: VIROL USTAV SAV,DUBRAVSKA CESTA 9/CS-84246 BRATISLAVA//CZECHOSLOVAKIA/ (Reprint); VYZKUMNY USTAV PREVENTIVNEJ & KLIN MED/CS-83301BRATISLAVA//CZECHOSLOVAKIA/; MESTSKA VET SPRAVA HL MESTA BRATISLAVY/CS-84213BRATISLAVA//CZECHOSLOVAKIA/
PUBLICATION: VETERINARNI MEDICINA, 1992, V37, N3, P177-183
GENUINE ARTICLE#: JG265
LANGUAGE: CZECH DOCUMENT TYPE: ARTICLE

ABSTRACT: The prevalence of Coxiella burnetii and Chlamydia psittaci antibodies was investigated in 530 dog specimens divided into six groups, i. e. A = private watch dogs, B1 = service dogs from Bratislava, B2 = service dogs froin other localities of Slovakia and Moravia, C = watch dogs from farms, I = household dogs, T = stray dogs. The dogs demonstrated the higher seropositivity to C. burnetii [11.7 %] than to Ch. psittaci [5.5 %]. The highest percentage of antibodies to C. burnetii was found in stray dogs [23.7 %], less prevalence of antibodies was observed in the animals in group C [13.6 %], almost the same positivity was proved in the dogs of group B1 and B2 [10.5 and 10.6 %]. The highest positivity to Ch. psittaci was demonstrated in the dogs of group A [8.7 %], less in group B2 [6.6 %]

and the least number in group B1 [1.9 %]. The stray dogs occupied the intermediate position in this data [Tab. I]. Ninety four localities were tested, from which 38 were seropositive. Neither acute coxiellosis nor chlamydiosis were proved in any animals examined. Ninety per cent of dogs were found healthy, but 10 % of dogs demonstrated hepatopathia and gastroenteritis. Two of them [category A and I] were seropositive to C. burnetii [titer 1 : 8 to 1 : 16] and one to Ch. psittaci [titer 1 : 16]. Both C. burnetii and Ch. psittaci attack dogs parallely with the agents of other zoonoses, of which the most common is Toxoplasma gondii [Tab. II]. Several dogs demonstrated seropositivity to three up to five zoonotic agents [Tab. III].

16/3,AB/29 (Item 9 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

02757721 References: 16
TITLE: THE SEROPREVALENCE OF *COXIELLOSIS"** (Q-FEVER) IN ONTARIO SHEEP FLOCKS
AUTHOR(S): LANG G; WALTNERTOEWS D; MENZIES P
CORPORATE SOURCE: UNIV GUELPH,ONTARIO VET COLL,DEPT VET MICROBIOL& IMMUNOL/GUELPH N1G 2W1/ONTARIO/CANADA/ (Reprint); UNIV GUELPH,ONTARIO VET COLL,DEPT POPULAT MED/GUELPH N1G 2W1/ONTARIO/CANADA/
PUBLICATION: CANADIAN JOURNAL OF VETERINARY RESEARCH-REVUE CANADIENNE DE RECHERCHE VETERINAIRE, 1991, V55, N2 (APR), P139-142
GENUINE ARTICLE#: FH317
LANGUAGE: ENGLISH DOCUMENT TYPE: ARTICLE

ABSTRACT: A serological survey for Coxiella burnetii was undertaken on a randomly selected population of 103 Ontario sheep flocks. Twenty-two flocks had at least one positive ewe; seven flocks had two or more reactors. The positive flocks were geographically clustered northwest of Guelph. Crutch-clipping of the ewe's wool prior to lambing, and total confinement housing at lambing in winter and spring seemed to lower the probability of seroreactivity of the flock ($p < 0.05$). The study suggests that sheep are not a major reservoir for Coxiella burnetii in Ontario.

16/3,AB/30 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0278773 DBA Accession No.: 2002-02914 PATENT
Polynucleotide construct encoding a processing component derived from N-terminal region of hepatitis virus ORF2 and an antigenic polypeptide useful for enhancing immune response to the polypeptide in an animal - plasmid-mediated sig1-2.1, sig2-2.1, sig3-3.1, glutathione-S-transferase fusion protein gene transfer and expression in mouse antigen presenting cell for gene therapy and nucleic acid vaccine
AUTHOR: Li F; Anderson D A; Purcell D F J
CORPORATE SOURCE: Fairfield, Victoria, Australia.
PATENT ASSIGNEE: Macfarlane-Burnet-Cent.Med.Res. 2001
PATENT NUMBER: WO 200173078 PATENT DATE: 20011004 WPI ACCESSION NO.: 2001-656919 (200175)
PRIORITY APPLIC. NO.: AU 20006616 APPLIC. DATE: 20000331
NATIONAL APPLIC. NO.: WO 2001AU353 APPLIC. DATE: 20010330

LANGUAGE: English
ABSTRACT: An isolated nucleic acid (I) encoding a fusion protein with a processing component (II, 108 or 150 bp DNA sequence encoding a 36 or 50 amino acid protein sequence) and an antigenic component (III), where (II) provides processing of (III) when (I) is expressed in a host cell and results in an enhancement of an immune response to (II) is claimed. Also claimed are: an isolated nucleic acid encoding (II); an a isolated antigen presenting cell transfected with (I); and a nucleic acid vaccine containing (I). Balb/C mice were inoculated with plasmid constructs or nucleic acid vaccines encoding fusion proteins of sig1-2.1, sig2-2.1 or sig3-3.1 derived from hepatitis E virus PORF2 protein, with glutathione-S-transferase (EC-2.5.1.18) and desired antigens via gene gun or i.m. injection. Sig1, sig2 and sig3 were derived by polymerase chain reaction(I) can be used for enhancing an immune response to a desired antigenic protein especially virus capsid protein in an animal. (I) can also be used for the production of medicine for human, e.g. fish or bird cancer, autoimmune diseases, virus, bacterium or parasitic infection prophylaxis and gene therapy. (47pp)

Set	Items	Description
S17	206	AU=(COWDEN, W? OR COWDEN W?)
S18	324	AU=(LAFFERTY, K? OR LAFFERTY K?)
S19	26	AU=(GAZDA, L? OR GAZDA L?)
S20	1	S17 AND S18 AND S19
S21	2	S17 AND (S18 OR S19)
S22	2	S17 AND (S18 OR S19)
S23	12	S18 AND S19
S24	542	S17 OR S18 OR S19
S25	4	S24 AND S3
S26	15	(S20 OR S21 OR S22 OR S23 OR S25) NOT (S9 OR S15)
S27	7	RD (unique items)

- Author(s)

>>>No matching display code(s) found in file(s): 65, 113

27/3,AB/1 (Item 1 from file: 77)
DIALOG(R)File 77:Conference Papers Index

4207853
Supplier Accession Number: 96-02368 V24N03
Antigen non-specific modulation of autoimmune diabetes in the nod mouse
Gazda, L.S.; Lafferty, K.J.
John Curtin Sch. Med. Res., Canberra, Australia
Immunology of Diabetes 1995 9545010 Location Unknown 31 Oct-3 Nov 1995
No sponsors listed.
International Publishers Distributor, 820 Town Center Drive, Langhorne, PA 19047. Paper No. A200

27/3,AB/2 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

13942043 Document Delivery Available: 000175629800015 References: 59
TITLE: Host systemic and local nitric oxide levels do not correlate with rejection of pig proislet xenografts in mice
AUTHOR(S): Simeonovic CJ (REPRINT); Cordery DV; Van Leeuwen B; Popp SK;

Townsend MJ; Paule MF; Wilson JD; *Cowden WB"**
AUTHOR(S) E-MAIL: Charmaine.Simeonovic@anu.edu.au
CORPORATE SOURCE: Australian Natl Univ, Div Mol Med, GPO Box 334/Canberra/ACT 2601/Australia/ (REPRINT); Australian Natl Univ, Div Mol Med, /Canberra/ACT 2601/Australia/; Australian Natl Univ, Div Cell Biol & Immunol, /Canberra/ACT 2601/Australia/; Australian Natl Univ, Div Biochem & Mol Biol, /Canberra/ACT 2601/Australia/; Canberra Hosp, Dept Endocrinol, /Woden/ACT/Australia/
PUBLICATION TYPE: JOURNAL
PUBLICATION: XENOTRANSPLANTATION, 2002, V9, N3 (MAY), P169-182
GENUINE ARTICLE#: 552PY
PUBLISHER: BLACKWELL MUNKSGAARD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK
ISSN: 0908-665X
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: The rejection of pig proislet xenografts in mice is a CD4 T cell-dependent process in which macrophages play an important role. To assess the potential for activated macrophages to act as effector cells in xenograft destruction, we have examined the relationship between proislet xenograft rejection, two principal markers of macrophage activation, transcription of inducible nitric oxide synthase (iNOS) and production of nitric oxide (NO), and their temporal relationship to intragraft cytokine gene expression. Xenograft rejection in CBA/H mice correlated with early induction of intragraft host iNOS mRNA and marked intragraft production of NO (reactive nitrogen intermediates, RNI). Intragraft mRNA expression for IFN-gamma, IL-1beta and TNF, cytokines associated with macrophage activation, was also found. These findings suggested that activated macrophages could be contributing to xenograft destruction via local NO-mediated toxicity at the graft site. To test the role of NO in this model: (1) *Q"**-*fever"** *antigen"** (*QFA"**) was administered to recipient mice in order to induce high systemic RNI levels and (2) in another experiment, pig proislets were transplanted into iNOS-/- mice. Treatment with *QFA"** correlated with prolonged xenograft survival at 7 days post-transplant. Splenocytes front *QFA"**-treated, but not control mice at 7 and 22 days post-transplant, exhibited inhibition of secondary xenogeneic mouse antiporcine mixed lymphocyte reaction (MLR) that was reversed by culture with the NOS inhibitor N-methylarginine (NMA). Despite continued elevated NO production, xenograft protection was temporary with complete rejection by day 22. Evidence that locally produced NO was not contributing to rejection was seen when pig proislets transplanted into iNOS-/- mice were rejected with normal kinetics; in these animals intragraft NO production was not detected (despite porcine iNOS gene expression). Failure of activated macrophages to achieve indefinite xenograft survival suggests that other factors are also required. Macrophage potential to effect either destructive or protective roles after pig proislet xenotransplantation suggests that such functions may depend on the site and magnitude of macrophage activation. Together these findings clearly demonstrate that high NO levels in the periphery are not damaging to xenogeneic islet tissue, neither host nor donor NO production is essential for islet xenograft rejection and consequently elevated plasma RNI levels do not represent a direct marker for rejection.

27/3,AB/3 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

09985362 References: 0
TITLE: Costimulation and the regulation of autoimmunity
AUTHOR(S): *Lafferty KJ (REPRINT)"**; *Gazda LS"**; Rose NR; Mackay IR
CORPORATE SOURCE: AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES/CANBERRA/ACT 2601/AUSTRALIA/ (REPRINT)
PUBLICATION TYPE: BOOK
PUBLICATION: AUTOIMMUNE DISEASES, THIRD EDITION, 1998, P59-74
GENUINE ARTICLE#: BL82S
PUBLISHER: ACADEMIC PRESS INC, 525 B STREET, SUITE 1900, SAN DIEGO, CA 92101-4495
ISBN: 0-12-596923-6 LIBRARY OF CONGRESS ID: 98-84368
LANGUAGE: English DOCUMENT TYPE: ARTICLE

27/3,AB/4 (Item 3 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

08569441 References: 47
TITLE: Diabetes results from a late change in the autoimmune response of NOD mice
AUTHOR(S): *Gazda LS"**; Charlton B; *Lafferty KJ (REPRINT)"**
CORPORATE SOURCE: AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES, DIV MOL MED, POB 334/CANBERRA/ACT 2601/AUSTRALIA/ (REPRINT); AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES, DIV MOL MED/CANBERRA/ACT 2601/AUSTRALIA/
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF AUTOIMMUNITY, 1997, V10, N3 (JUN), P261-270
GENUINE ARTICLE#: XG033
PUBLISHER: ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON, ENGLAND NW1 7DX
ISSN: 0896-8411
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: IDDM in the NOD mouse is the result of a chronic autoimmune process. NOD mice are shown to express benign autoimmunity that converts to a state of malignant autoimmunity and the development of IDDM. Young disease-prone NOD mice are in a state of benign autoimmunity that is correlated with a non-destructive response to islet tissue and the preservation of insulin-containing beta-cells. A proportion of mice with benign autoimmunity convert to having malignant autoimmunity. Clinical diabetes is diagnosed approximately 3 weeks from the development of malignant autoimmunity which is correlated with a destructive response to grafted islet tissue and extensive beta-cell destruction. We conclude that the development of clinical disease is correlated with a change in the state of autoimmunity, that is, from benign to malignant autoimmunity. (C) 1997 Academic Press Limited.

27/3,AB/5 (Item 4 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

08311196 References: 45
TITLE: Tolerance: Case of self/not-self discrimination maintained by clonal deletion?
AUTHOR(S): *Lafferty KJ (REPRINT)"**; *Gazda LS"**
CORPORATE SOURCE: AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES, DIV MOL MED, POB 334/CANBERRA/ACT 2601/AUSTRALIA/ (REPRINT); AUSTRALIAN NATL UNIV,/CANBERRA/ACT/AUSTRALIA/

PUBLICATION TYPE: JOURNAL
PUBLICATION: HUMAN IMMUNOLOGY, 1997, V52, N2 (FEB), P119-126
GENUINE ARTICLE#: WQ242
PUBLISHER: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
ISSN: 0198-8859
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: The clonal selection theory of immune reactivity is based on a metaphor of self/not-self discrimination and considers self-tolerance to result from clonal deletion. There is evidence that a deletional mechanism is responsible for negative selection of self MHC-reactive T-cells in the thymus. Bretscher/Cohn cheery builds on this concept and provides a model which allows self/not-self discrimination to occur at any time throughout che life of the individual. However, modern concepts of antigen presentation in which MHC-peptide co-presentation is the unit recognised by the T-cell receptor have abandoned the Bretscher/Cohn requirement for associative recognition of antigen. For this reason, such models of APC function cannot use Bretscher/Cohn theory to explain self/not-self discrimination. Matzinger's 'danger' metaphor for the immune system provides a theoretical way forward by moving the emphasis away from an immune system based on self/not-self discrimination. These theoretical developments lead to a novel approach to the control of autoimmunity that is based on the strengthening of immune regulation by the use of adjuvant therapy. (C) American Society for Histocompatibility and Immunogenetics, 1997.

27/3,AB/6 (Item 5 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

07870943 References: 33
TITLE: Regulation of autoimmune diabetes: Characteristics of non-islet-antigen specific therapies
AUTHOR(S): *Gazda LS"**; Baxter AG; *Lafferty KJ"**
CORPORATE SOURCE: AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES, DIV MOL MED/CANBERRA/ACT 2601/AUSTRALIA/ (REPRINT); AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES, DIV MOL MED/CANBERRA/ACT 2601/AUSTRALIA/; CENTENARY INST CANC MED & CELL BIOL,/NEWTOWN/NSW/AUSTRALIA/
PUBLICATION TYPE: JOURNAL
PUBLICATION: IMMUNOLOGY AND CELL BIOLOGY, 1996, V74, N5 (OCT), P401-407
GENUINE ARTICLE#: VQ232
PUBLISHER: BLACKWELL SCIENCE, 54 UNIVERSITY ST, P O BOX 378, CARLTON VICTORIA 3053, AUSTRALIA
ISSN: 0818-9641
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Non-islet-antigen specific treatments have been shown to alter the naturalhistory of insulin dependent diabetes in both the non-obese diabetic (NOD)mouse and in recently diagnosed patients. However, concerns have been raised regarding the possibility that non-islet-antigen specific therapy may trade cell mediated autoimmunity for antibody dependent autoimmunity. Female NOD mice at approximately 70 days of age were treated with the non-islet-antigen specific agents complete Freund's adjuvant (CFA) and Bacillus Calmette-Guerin (BCG) and assayed for the development of antibody mediated autoimmunity at 300 days of age. Autoantibodies to red cells were not detected in any of the BCG (n = 19) or CFA (n = 15) treated

animals, while 2 of 13 age-matched NOD animals had autoantibodies to red cells, shown by a positive direct Coomb's test. Anti-nuclear autoantibodies and complement deposition inthe renal glomeruli were not significantly increased in the treated animals as compared to age-matched non-diabetic mice. The relative effectiveness of CFA and BCG treatment was examined in terms of the ability of these agents to preserve insulin containing islets. Complete Freund's adjuvant treatment was found to be more effective in preserving insulin containing islets when compared to BCG treatment. This study demonstrates that it is possibleto inhibit the development of autoimmune diabetes without increasing the probability that treated animals will develop antibody dependent autoimmunity.

27/3,AB/7 (Item 6 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

07082806 References: 14
TITLE: AUTOIMMUNE DIABETES - CAUGHT IN THE CAUSALITY TRAP
AUTHOR(S): *GAZDA LS"**; GILCHRIST KA; *LAFFERTY KJ (Reprint)"**
CORPORATE SOURCE: AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES,POB 334/CANBERRA/ACT 2601/AUSTRALIA/ (Reprint); AUSTRALIAN NATL UNIV,JOHN CURTIN SCH MED RES/CANBERRA/ACT 2601/AUSTRALIA/
PUBLICATION: IMMUNOLOGY AND CELL BIOLOGY, 1995, V73, N6 (DEC), P549-551
GENUINE ARTICLE#: TR612
ISSN: 0818-9641
LANGUAGE: ENGLISH DOCUMENT TYPE: ARTICLE

ABSTRACT: The lack of concordance between genotype and clinical diabetes has prompted a search for the infectious agent that precipitates this autoimmune disease. However, this approach may be misleading. It assumes that the disease-prone individuals that do not develop diabetes do not have autoimmunity. In the non-obese diabetic (NOD) mouse, the genotype is a primary determinant of autoimmunity. Not all animals of the disease-prone genotype develop clinical disease; however, all have autoimmunity. This is expressed as a destructive or non-destructive process. Multiple pathways are open to the immune system and whether or not the immune response is destructive and leads to the development of clinical disease, appears to be a random process. If this is the case, the most important questions relating to autoimmune disease are not those concerning the 'causative' agents. Instead we should be asking what are the differences between pathways open to the immune system and what factors affect the probability that one or another pathway is finally selected?
? log y

Devi, S.
09/887773

(FILE 'HCAPLUS' ENTERED AT 11:03:34 ON 07 JUN 2002)
L1 1628 SEA FILE=HCAPLUS ABB=ON PLU=ON COXIELLA OR BURNETII OR (Q FEVER OR QF)(W)ANTIGEN OR QFA OR ANTIGEN?(W)COMPONENT
L2 15 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 AND ((AUTOIMMUN? OR AUTO IMMUN?)(5A)(DISEAS? OR DISORDER) OR COXIELLOS? OR DIABET? OR IDDM)

-key terms

L2 ANSWER 1 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:418396 HCAPLUS
TITLE: Mouse resident peritoneal macrophages partially control in vitro infection with **Coxiella burnetii** phase II
AUTHOR(S): Zamboni, Dario S.; Mortara, Renato A.; Freymuller, Edna; Rabinovitch, Michel
CORPORATE SOURCE: 6o andar, Rua Botucatu 862, UNIFESP, Escola Paulista de Medicina, Disciplina de Parasitologia, Imunologia e Parasitologia, Departamento de Microbiologia, SP, Sa o Paulo, 04023-062, Brazil
SOURCE: Microbes and Infection (2002), 4(6), 591-598
CODEN: MCINFS; ISSN: 1286-4579
PUBLISHER: Editions Scientifiques et Medicales Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Coxiella burnetii**, the agent of Q fever in man and of **coxiellosis** in other species, is a small, dimorphic, obligate intracellular bacterium, sheltered within large, acidified, and hydrolase-rich phagosomes. Although several primary and established cell lines, macrophage-like cells, and primary macrophages from other species have been infected with C. **burnetii**, the infection of mouse primary macrophages has not been sufficiently characterized. In this report quantification of DAPI (4', 6-diamino-2-phenylindole) fluorescence images acquired by confocal microscopy, and transmission electron microscopy were used to compare the infection of three mouse-derived cells, L929 fibroblasts, J774 macrophage-like cells, and resident peritoneal macrophages, with a phase II clone of C. **burnetii** known to be non-virulent for mammals. Infected peritoneal phagocytes differed from L929 or J774 cells in that: (a) large vacuoles took longer to appear (3-5 d instead of 2), and were only found in a subset (20-30%) of macrophages, as opposed to in more than 70% of the other cells; (b) total and vacuole-assocd. relative bacterial loads in L929 and J774 cells were several-fold higher than in peritoneal macrophages; (c) estd. doubling times of the bacteria were about 68 h in the primary macrophages, 18 h in J774 and 22 h in L929 cells. Thus, mouse resident peritoneal macrophages control both the formation of the large vacuoles and the intracellular proliferation of C. **burnetii** phase II.

L2 ANSWER 2 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:564860 HCAPLUS
DOCUMENT NUMBER: 135:142203
TITLE: Vaccine composition for prevention of pathogen-induced infections and malignant diseases and immune disorders
INVENTOR(S): Jira, Vic; Jirathitical, Vichai
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 56 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001054717	A1	20010802	WO 2001-US2811	20010129

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-494607 A 20000131
US 2000-227520P P 20000824

AB A vaccine compn. for treating or preventing pathogen-induced infections, malignant diseases, and immune **disorders**, i.e., inflammation and **autoimmune diseases**, is disclosed, along with a process for manufg. the compn. and various methods of using the compn. The compn. comprises pathogen-infected cell or tissue, or malignantly or immunol. aberrant cells or tissues which are reduced and/or denatured. The preferred compn. is administered across the mucosal surface of a subject suffering or about to suffer from infection, tumor, or immune disease. The compn. is administered as a preventive or a therapeutic vaccine.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:742368 HCAPLUS
DOCUMENT NUMBER: 133:308998
TITLE: Method for judging **autoimmune disease**, method for detecting anti-Reg protein autoantibody and diagnostics for **autoimmune diseases**
INVENTOR(S): Okamoto, Hiroshi
PATENT ASSIGNEE(S): Hitachi Chemical Co., Ltd., Japan
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062066	A1	20001019	WO 2000-JP2245	20000406

W: AU, CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

EP 1167974 A1 20020102 EP 2000-915389 20000406
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRIORITY APPLN. INFO.: JP 1999-99963 A 19990407
WO 2000-JP2245 W 20000406

AB A method for judging an **autoimmune disease** characterized by detecting the existence of anti-Reg protein autoantibody in a specimen; and a method for judging insulin dependent or noninsulin-dependent **diabetes**. A method for detecting anti-Reg protein autoantibody characterized by bringing a specimen into contact with an **antigen component** and detecting the formation of an immune complex. Diagnostics for **autoimmune diseases** which contain an **antigen component** capable of binding specifically to anti-Reg protein autoantibody; and diagnostics for insulin-dependent or noninsulin-dependent **diabetes**.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:251660 HCAPLUS
DOCUMENT NUMBER: 133:251037
TITLE: Distribution of immunoglobulin G (IgG) and A (IgA) subclasses following Q fever vaccination with soluble phase I **Coxiella burnetii** extract
AUTHOR(S): Camacho, M. T.; Outschoorn, I.; Kovacova, E.; Tellez, A.
CORPORATE SOURCE: CNM, Instituto de Salud Carlos III, Madrid, Spain
SOURCE: Vaccine (2000), 18(17), 1773-1777
CODEN: VACCDE; ISSN: 0264-410X
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB High levels of IgG1, IgG3 and IgA2 antibodies have been obsd. in patients with Q fever following **Coxiella burnetii** infection. This IgG subclass distribution is more typical of viral and **autoimmune diseases** than of bacterial infections. It seemed, therefore, of interest to carry out a prospective study of the distribution of Ig subclasses after vaccination with phase 1 C. **burnetii** tricloroacetic sol. exts. to detect possible differences with respect to natural infection. The antibody response found in vaccinees was mainly restricted to the IgG1, IgG2 and IgA1 subclasses. These findings confirm differences in isotype distribution when compared to those of patients with acute or chronic **Coxiella** infections and opens an area of interest with respect to the role of IgA subclasses.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:86637 HCAPLUS
DOCUMENT NUMBER: 133:57423
TITLE: Circulating anticentromere CENP-A and CENP-B

antibodies in patients with diffuse and limited systemic sclerosis, systemic lupus erythematosus, and rheumatoid arthritis

AUTHOR(S): Russo, Katherine; Hoch, Sallie; Dima, Corina; Varga, John; Teodorescu, Marius

CORPORATE SOURCE: Department of Microbiology/Immunology, Section of Rheumatology, College of Medicine, University of Illinois at Chicago, USA

SOURCE: Journal of Rheumatology (2000), 27(1), 142-148
CODEN: JRHUA9; ISSN: 0315-162X

PUBLISHER: Journal of Rheumatology Publishing Co. Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Purpose: To det. the disease sensitivity and specificity of testing for autoantibodies against 2 of the 3 main human centromere **antigenic components**, CENP-A and CENP-B (recombinant, expressed in baculovirus). ELISA with CENP-A and CENP-B antigens were used to test 45 sera showing a centromere pattern by immunofluorescence (IFA) and sera from 96 patients with systemic sclerosis (SSc), subdivided into diffuse (dSSc) and limited (lSSc) forms. For controls, the same tests were performed on sera from 100 patients with rheumatoid arthritis (RA), 100 with systemic lupus erythematosus (SLE), and 50 random blood donors. Sera from all the patients with SSc were also tested for the presence of anti-Sc170 antibody by ELISA (bovine antigen), and for pattern and titer by IFA (HEp-2 cells). Of the 45 IFA pos. sera, 93% were pos. for anti-CENP-A and 91% for anti-CENP-B. There was a very good quant. correlation between the antibody levels against these 2 centromere components ($r = 0.597$; $p < 0.001$). Anti-CENP-A and B were found in 48% of patients with lSSc, and in 11% and 9%, resp., of those with dSSc. The difference in the frequency of anti-CENP-A between the 2 patient groups was significant (chi-squared, $p < 0.001$). Similar levels of anticentromere staining pattern by IFA were obsd. for these 2 groups. Anti-Sc170 was elevated in 8% of lSSc and 25% of dSSc patients; this difference was also significant (chi-squared, $p = 0.02$). Neither CENP-A nor CENP-B reacted with IgG from SSc patients contg. anti-Sc170. The frequency of abnormal levels in patients with SLE and RA was, resp., 11% and 3% for anti-CENP-A and 4% and 3% for anti-CENP-B. The reaction of IgG from SLE and RA patients with CENP-A was not inhibited by histone H3, i.e., it was not due to recognition of the histone-like domain in CENP-A. Thus, when 96 SSc patients were compared to 200 patients with RA and SLE, the disease specificity of anti-CENP-A and B was 93% and 96.5%, resp. In addn. to IFA, ELISA tests for CENP-A and CENP-B yield results with similar sensitivity and specificity for the diagnosis of SSc. CENP-A and CENP-B are primarily assocd. with lSSc. In SSc the autoantibody response is directed simultaneously and with similar amplitude against these 2 components of the centromere structure, whereas in other **autoimmune diseases** the response is directed mainly against one of the 2 components.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:531885 HCAPLUS
DOCUMENT NUMBER: 131:196604

TITLE: Detection of anti-nuclear antibodies in liver diseases by indirect immunofluorescence method and enzyme-linked immunosorbent assay
AUTHOR(S): Koizumi, Hideko; Onozuka, Yasushi; Shibata, Minoru; Sano, Kiniti; Oosima, Yasuo; Miyachi, Kiyomitu; Ueno, Yukihisa
CORPORATE SOURCE: Dep. Clin. Lab., Kawasaki Cent. Hosp., Kawasaki, 210-0822, Japan
SOURCE: Rinsho Byori (1999), 47(8), 744-748
CODEN: RBYOAI; ISSN: 0047-1860
PUBLISHER: Rinsho Byori Gakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Detection of anti-nuclear antibodies (ANA) is essential for diagnosing **autoimmune diseases** including **autoimmune** liver **diseases**. An indirect immunofluorescence (IIF) method with a cell line (HEp-2) derived from human laryngeal carcinoma has been used as a std. substrate. Recently, an ELISA (ELISA) using multiple solid-phase antigens has been developed. We assayed sera from 272 cases of chronic liver diseases, 91 cases of healthy subjects and studied clin. significance of ANA. The sensitivity of IIF method in detection of ANA (fluorescence-ANA : FANA) and that of ELISA (ELISA-ANA : EANA) were 19.2% and 17.3%, in chronic hepatitis B(CH-B), 16.7%, and 17.3%, in chronic hepatitis C(CH-C), 84.2% and 50.9% in primary biliary cirrhosis (PBC), 100%, and 85.7%, in autoimmune hepatitis (AIH) and 15.4%, and 18.7% in healthy subjects. The sensitivity of EANA was considerably lower than that of FANA in PBC and AIH, but the sensitivity was the same in CH-C, CH-B, and healthy subjects. Because the solid-phase target antigens do not include nuclear **antigen components** recognized only by patients with PBC or AIH, ELISA can not detect all the species of ANA This accounts for the low sensitivity of EANA in PBC and AIH. In conclusion, the current EANA is useful for screening of ANA, but FANA should be performed when PBC or AIH is suspected.

L2 ANSWER 7 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:284642 HCAPLUS
DOCUMENT NUMBER: 129:94336
TITLE: Immunological reactivity of **diabetes** -prone BB/OK rats to syngeneic antigens: effect on .beta.-cell destruction and **diabetes** onset
AUTHOR(S): Schroder, Dieter; Schmidt, Siegfried; Kloting, Ingrid; Honig, Anke; Lucke, Silke; Hehmke, Bernd; Schlosser, Michael
CORPORATE SOURCE: Institute of Diabetes "Gerhardt Katsch", Ernst-Moritz-Arndt-University, Karlsburg, D-17495, Germany
SOURCE: Advances in Experimental Medicine and Biology (1997), 426(Physiology and Pathophysiology of the Islets of Langerhans), 345-353
CODEN: AEMBAP; ISSN: 0065-2598
PUBLISHER: Plenum Publishing Corp.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Young BB/OK rats exhibit complement-dependent antibody-mediated cytotoxicity (C'AMC) to islet cells as well as to pancreatic

exocrine cells but only 50-60% of the animals become **diabetic**. However, this does not exclude that C'AMC is involved in .beta.-cell alteration in vivo. It might be that in young BB/OK rats only weak lesions of islet cells or adjacent non-endocrine tissue are caused by C'AMC but liberated **antigenic components** subsequently initiate inflammatory processes thus being of importance for the extent of .beta.-cell destruction. This study compared the immunol. reactivity of **diabetes**-prone BB/OK rats in response to intrasplenic application of syngeneic antigens by measuring the appearance of C'AMC to islet cells, exocrine cells, and lymphocytes and the impact on **diabetes** development.

L2 ANSWER 8 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:146381 HCAPLUS
DOCUMENT NUMBER: 126:198401
TITLE: Alcohol dehydrogenase: a target of humoral **autoimmune** response in liver **disease**
AUTHOR(S): Ma, Yun; Gaken, Joop; McFarlane, Barbara M.; Foss, Yvonne; Farzaneh, Farzin; McFarlane, Ian G.; Mieli-Vergani, Giorgina; Vergani, Diego
CORPORATE SOURCE: Inst. Heptaol., Univ. Coll. London Med. Sch., London, UK
SOURCE: Gastroenterology (1997), 112(2), 483-492
CODEN: GASTAB; ISSN: 0016-5085
PUBLISHER: Saunders
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Liver-specific membrane lipoprotein (LSP) is a heterogeneous liver prepn. that has been widely used to study autoreactivity in liver disease. The aim of this study was to identify autoantigens in LSP. Guinea pig anti-LSP serum was used to screen a human liver cDNA library. Humoral immune responses to isolated potential autoantigens were investigated by immunoblotting in 91 pediatric patients with various liver diseases, 20 adult patients with alc. liver **disease** and 20 with **autoimmune** thyroid **disease**, 37 healthy children, and 20 healthy adults. A 1.6-kilobase cDNA insert isolated from the cDNA library was found to encode amino acids 61-374 of the human alc. dehydrogenase (ADH)-.gamma.1 subunit. Antibodies to this or other ADH subunits were found significantly more frequently in **autoimmune** liver **diseases** (19 of 39 patients; 49%), Wilson's disease (5 of 13 patients; 38%), and alc. liver disease (10 of 20 patients; 50%) than in normal controls and correlated with **disease** activity in **autoimmune** liver **disease**. ADH has been identified as a new **antigenic component** of the LSP using a xenogeneic antiserum to immunoprobe a human cDNA liver library and seems to be a target autoantigen in liver disease. This approach may be useful in identifying other potential autoantigens.

L2 ANSWER 9 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:698763 HCAPLUS
DOCUMENT NUMBER: 121:298763
TITLE: Immunohistochemical characterization of monoclonal antibodies directed against the TSH receptor

AUTHOR(S): Kohnert, Klaus-Dieter; Krabbe, Siegfried; Meng, Wieland
CORPORATE SOURCE: Institute Diabetes "Gerhardt Katsch", University Greifswald, Karlsburg/Greifswald, 17495, Germany
SOURCE: Acta Histochem. (1994), 96(2), 175-80
CODEN: AHISA9; ISSN: 0065-1281
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Monoclonal antibodies have been obtained by fusing mouse myeloma cells with spleen cells of mice immunized with crude thyroid membranes. Among the antibodies reactive with different thyroid **antigenic components**, 3 were found to specifically react with TSH receptor mols. These antibodies displayed characteristic staining patterns on frozen sections of thyroid tissue from patients with various thyroid diseases upon identification of antibody binding by indirect peroxidase staining. No specific reactivity was detected with tissue from other human organs, such as pancreas, liver, fat, and muscle. The results demonstrate that the immunoperoxidase technique and the specificity of the monoclonal antibodies produced permitted the identification of cellular constituents that might be important antigens in **autoimmune** thyroid **disease**.

L2 ANSWER 10 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:58120 HCAPLUS
DOCUMENT NUMBER: 118:58120
TITLE: MHC conjugates useful in ameliorating autoimmunity
INVENTOR(S): Clark, Brian R.; Sharma, Somesh D.; Lerch, Bernard L.
PATENT ASSIGNEE(S): Anergen, Inc., USA
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9218150	A1	19921029	WO 1992-US3391	19920423
W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, AU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO				
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GN, GR, IT, LU, MC, ML, MR, NL, SE				
US 5260422	A	19931109	US 1991-690840	19910423
AU 9219144	A1	19921117	AU 1992-19144	19920423
JP 06507168	T2	19940811	JP 1992-511424	19920423
EP 630255	A1	19941228	EP 1992-911380	19920423
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE				

PRIORITY APPLN. INFO.:
US 1991-690840 A 19910423
US 1988-210594 B1 19880623
US 1990-576084 A2 19900830
US 1992-869293 A 19920414
WO 1992-US3391 A 19920423

AB Complexes consisting of an isolated MHC **antigen component** and an (auto)antigenic peptide assocd. with the antigen binding site of the MHC component are presented which are

useful for treating **autoimmune diseases**. Anergy was induced in T-cells derived from the thymus of a young-onset myasthenia gravis patient by preincubating the T-cells with HLA-DR4Dw4 antigen complexed with peptide 138-167 of acetylcholine receptor .alpha.-subunit.

L2 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:551321 HCAPLUS
DOCUMENT NUMBER: 105:151321
TITLE: Scl 70 autoantibodies from scleroderma patients recognize a 95 kDa protein identified as DNA topoisomerase I
AUTHOR(S): Guldner, Hans Herbert; Szostecki, Carin; Vosberg, Hans Peter; Lakomek, Heinz Juergen; Penner, Edward; Bautz, Friedlinde A.
CORPORATE SOURCE: Inst. Mol. Genet., Univ. Heidelberg, Heidelberg, D-6900, Fed. Rep. Ger.
SOURCE: Chromosoma (1986), 94(2), 132-8
CODEN: CHROAU; ISSN: 0009-5915
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sera of patients suffering from the **autoimmune disease** progressive systemic sclerosis (PSS) are known to contain autoantibodies which have been reported to recognize a 70 kilodalton (kDa) antigenic protein, designated the Scl 70 antigen. By immunoblotting of nuclear exts. from HeLa cells with sera from scleroderma patients it was obsd. that the size of the antigen present in such cells depends on the conditions of antigen isolation. When protease inhibitors were included in the extn. buffer, a 95 kDa protein was identified instead of a 70 kDa protein. When protease inhibitors were omitted, a no. of polypeptides in the size range 66 to 95 kDa was found. Antibodies which had been affinity purified on the 95 kDa antigen cross-reacted with the 66-95 kDa polypeptides. These results suggest that the smaller proteins were degrdn. products of the 95 kDa antigen. Immunofluorescence studied on PtK-2 cells with the antibody specific for the 95 kDa protein gave staining of nuclei, nucleoli and of chromosomes and the nuclear organizer region in mitotic cells. Since this distribution of antigens within the nucleus was reminiscent of the intranuclear distribution of DNA topoisomerase I found by others, the authors probed purified DNA topoisomerase I from calf thymus directly with the autoantibodies from PSS patients, and also the 95 kDa antigens of HeLa cell nuclei with antibodies raised against the bovine DNA topoisomerase I. From the cross-reaction pattern obsd. with the different antigens and antibodies it was concluded that DNA topoisomerase I is one of the **antigenic components** against which autoantibodies are formed in scleroderma patients.

L2 ANSWER 12 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:468664 HCAPLUS
DOCUMENT NUMBER: 99:68664
TITLE: Isolation and quantitation of immune complexes in **diabetic** Syrian hamsters: a chronological study
AUTHOR(S): McDonald, Thomas L.; Quenette, Linda; Phares, C. K.
CORPORATE SOURCE: Med. Cent., Univ. Nebraska, Omaha, NE, 68105, USA

SOURCE: J. Clin. Lab. Immunol. (1983), 11(3), 129-33
CODEN: JLIMDJ; ISSN: 0141-2760
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Immune complexes (IC) were isolated from the serum of streptozotocin (Sz)-induced **diabetic** hamsters by co-pptn. with an equine rheumatoid-complement C1q factor. The IC, comprised of an unknown antigen(s) and IgG, occurred at 2 distinct time intervals during the 12-wk chronol. study. Although all **diabetic** hamsters with hyperglycemia of 300 mg/kL had IC, there was no correlation between the occurrence or concn. of IC and the degree of hyperglycemia. The cycling nature of IC in the serum of **diabetic** hamsters suggests that the **antigen component** either fluctuates chronol. or that the IC contain different antigens that do not occur simultaneously.

L2 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:525423 HCAPLUS
DOCUMENT NUMBER: 97:125423
TITLE: Analysis of speckled fluorescent antinuclear antibody test antiserums using electrofocused nuclear antigens
AUTHOR(S): Okarma, Thomas B.; Krueger, Judith Ann; Holman, Halsted R.
CORPORATE SOURCE: Sch. Med., Stanford Univ., Stanford, CA, 94305, USA
SOURCE: J. Clin. Invest. (1982), 70(2), 296-303
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Antibodies to different components of the extractable nuclear antigen (ENA) have been thought to be serol. markers for clin. subsets of rheumatic diseases. However, incomplete characterization and standardization of **antigenic components** such as ribonucleoprotein (RNP), Sm, and SS-B (Ha), and the multiplicity of autoantibodies produced by different patients have confounded correlations between autoantibody specificity and disease subsets. The preparative sepn. of the antigens Sm, RNP, and SS-B (Ha) by electrofocusing and their use in a rocket electrophoretic assay is described. The method, in one step, identifies and quantifies the multiple reactivities of patient sera exhibiting the speckled fluorescent antinuclear antibody test (FANA) pattern. Preparative electrofocusing generates milligram quantities of these antigens with retention of their immunol. and biochem. characteristics, facilitating further study of their biol. properties and relationships to disease subsets.

L2 ANSWER 14 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1971:474133 HCAPLUS
DOCUMENT NUMBER: 75:74133
TITLE: Antigens and autoantigens of the seminal vesicle. I. Immunochemical studies on guinea pig vesicular fluid
AUTHOR(S): Orsini, Frank; Shulman, Sidney
CORPORATE SOURCE: Dep. Microbiol., New York Med. Coll., New York, N. Y., USA
SOURCE: J. Exp. Med. (1971), 134(1), 120-40
CODEN: JEMEAV

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Guinea pig vesicular fluid was characterized biochem. and immunol. This fluid was homogeneous on ultracentrifugal anal., revealing a single boundary with a sedimentation coeff. of 1.5 S. Electrophoretic sepn. methods revealed 6 components, of which 3 were major and of approx. equal proportions. They were termed I, II and III. The II component was strongly antigenic in heteroimmunization, whereas components I and III failed to show any antigenicity. This **antigen** (**component** II) was highly tissue specific and species specific. Through procedures of isoimmunization component II was found to be immunogenic, giving rise (in male animals) to autoantibodies. A high proportion of injected guinea pigs showed pos. skin tests and many revealed tissue lesions when the seminal vesicles were examd. histol. It is concluded that exptl. **autoimmune disease** of the seminal vesicle has been induced.

L2 ANSWER 15 OF 15 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1971:40446 HCAPLUS
DOCUMENT NUMBER: 74:40446
TITLE: Dysproteinemia in **diabetes** mellitus and its role in the pathogenesis of **diabetic** angiopathies
AUTHOR(S): Efimov, A. S.; Bodnar, P. N.; Limanskaya, G. F.; Lapko, L. I.; Kopytov, Yu. P.
CORPORATE SOURCE: Kiev. Inst. Endokrinol. Obmena Veshchestv, Kiev, USSR
SOURCE: Probl. Endokrinol. (1970), 16(6), 7-11
CODEN: PROEAS
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The protein, glycoprotein, and lipoprotein fractions were detd. in **diabetics** by filter paper electrophoresis. **Antigen components** of blood serum were detd. by immunoelectrophoresis, and the binding properties of transcortin were estd. Decreased values of albumin and increased values of the .alpha.-globulin fraction were found. Increased values of glycoproteins and mucoproteins were also obsd. The .gamma.-globulin fraction was lower in all cases. The .beta.-lipoprotein fraction was increased according to the seriousness of the angiopathies regardless of whether the changes in the arterial wall had an org. or functional character only. In the immunoelectrophoresis, increased nos. of pptn. lines were obsd., with high intensities for the pptn. lines of the immunoglobulins, ceruloplasmin, and .alpha.-globulins. The simultaneous detns. of 11-hydroxy steroids and transcortin binding capacity showed increased values of the former and decreased values of the latter. These values also depended on the seriousness of the **diabetes**.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO, VETU, VETB, CABA, AGRICOLA, PHIC, PHIN, TOXCENTER' ENTERED AT 11:10:25 ON 07 JUN 2002)
L3 190 S L2
L4 111 DUP REM L3 (79 DUPLICATES REMOVED)
L5 26 S L4 AND (TREAT? OR THERAP?)

L5 ANSWER 1 OF 26 MEDLINE

ACCESSION NUMBER: 2002303544 IN-PROCESS
DOCUMENT NUMBER: 22039960 PubMed ID: 12045166
TITLE: Celiac **disease** associated with **autoimmune** myocarditis.
AUTHOR: Frustaci Andrea; Cuoco Lucio; Chimenti Cristina; Pieroni Maurizio; Fioravanti Giuseppina; Gentiloni Nicola; Maseri Attilio; Gasbarrini Giovanni
CORPORATE SOURCE: Departments of Cardiology (A.F., C.C., M.P., A.M.), Internal Medicine (L.C., N.G., G.G.), and Transplant Surgery (F.G.), Catholic University, Rome, Italy.
SOURCE: CIRCULATION, (2002 Jun 4) 105 (22) 2611-8. Journal code: 0147763. ISSN: 1524-4539.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals
ENTRY DATE: Entered STN: 20020605
Last Updated on STN: 20020605

AB BACKGROUND: Both celiac disease (CD) and myocarditis can be associated with systemic **autoimmune disorders**; however, the coexistence of the 2 entities has never been investigated, although its identification may have a clinical impact. Methods and Results- We screened the serum of 187 consecutive patients with myocarditis (118 males and 69 females, mean age 41.7+/-14.3 years) for the presence of cardiac autoantibodies, anti-tissue transglutaminase (IgA-tTG), and anti-endomysial antibodies (AEAs). IgA-tTG-positive and AEA-positive patients underwent duodenal endoscopy and biopsy and HLA analysis. Thirteen of the 187 patients were positive for IgA-tTG, and 9 (4.4%) of them were positive for AEA. These 9 patients had iron-deficient anemia and exhibited duodenal endoscopic and histological evidence of CD. CD was observed in 1 (0.3%) of 306 normal controls (P<0.003). In CD patients, myocarditis was associated with heart failure in 5 patients and with ventricular arrhythmias (Lown class III-IVa) in 4 patients. From histological examination, a lymphocytic infiltrate was determined to be present in 8 patients, and giant cell myocarditis was found in 1 patient; circulating cardiac autoantibodies were positive and myocardial viral genomes were negative in all patients. HLA of the patients with CD and myocarditis was DQ2-DR3 in 8 patients and DQ2-DR5(11)/DR7 in 1 patient. The 5 patients with myocarditis and heart failure received immunosuppression and a gluten-free diet, which elicited recovery of cardiac volumes and function. The 4 patients with arrhythmia, after being put on a gluten-free diet alone, showed improvement in the arrhythmia (Lown class I). CONCLUSIONS: A common autoimmune process toward **antigenic components** of the myocardium and small bowel can be found in >4% of the patients with myocarditis. In these patients, immunosuppression and a gluten-free diet can be effective **therapeutic** options.

L5 ANSWER 2 OF 26 MEDLINE
ACCESSION NUMBER: 2001261672 MEDLINE
DOCUMENT NUMBER: 21199702 PubMed ID: 11303304
TITLE: Anti-nuclear envelope antibodies: Clinical associations.
AUTHOR: Nesher G; Margalit R; Ashkenazi Y J
CORPORATE SOURCE: Department of Rheumatology Service, Hebrew University

Medical School, Jerusalem, Israel..
nesher@inter.net.il
SOURCE: SEMINARS IN ARTHRITIS AND RHEUMATISM, (2001 Apr) 30 (5) 313-20. Ref: 42
Journal code: UMV; 1306053. ISSN: 0049-0172.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW OF REPORTED CASES)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

AB OBJECTIVES: Characterization of the clinical associations and clinical implications of antibodies reacting with antigens of the nuclear envelope. METHODS: Description of an illustrative case and a MEDLINE search-assisted literature review of relevant cases. RESULTS: With indirect immunofluorescence, autoantibodies directed against various antigens of the nuclear envelope stain the nucleus in a ring-like (rim) pattern. Autoantibodies against 5 **antigenic components** of the nuclear envelope have been described: anti-gp210, p62, lamina, lamina-associated polypeptides, and lamin B receptor. Antibodies to antigens of the nuclear pore complex, such as gp210 and p62, are highly specific (> 95%) for primary biliary cirrhosis and may aid in the serologic diagnosis of this condition, especially in cases in which antimitochondrial antibodies are not detectable. In contrast, antilamin antibodies are not disease-specific but seem to be associated with lupus anticoagulant or anticardiolipin antibodies, antiphospholipid syndrome, thrombocytopenia, **autoimmune** liver **diseases**, and arthralgia. High-titered antilamin antibodies help to define a subset of lupus patients with antiphospholipid antibodies who are at a lower risk of developing thrombotic events. In addition, preliminary data suggest that the presence of antilamin antibodies may be helpful in the diagnosis of chronic fatigue syndrome. CONCLUSIONS: Each of the antibodies reacting with nuclear membrane antigens has its own spectrum of disease associations. RELEVANCE: Determination of anti-nuclear envelope antibody pattern by indirect immunofluorescence, with subsequent determination of the specific antibody, carries important diagnostic and prognostic implications in various autoimmune conditions.

L5 ANSWER 3 OF 26 MEDLINE
ACCESSION NUMBER: 2001091485 MEDLINE
DOCUMENT NUMBER: 21022387 PubMed ID: 11141638
TITLE: Vogt-Koyanagi-Harada disease.
AUTHOR: Read R W; Rao N A; Cunningham E T
CORPORATE SOURCE: Department of Ophthalmology, Doheny Eye Institute, Pathology, University of Alabama at Birmingham, Birmingham, 700 18th Street South, EFH 6001 Alabama, USA.. rwr@uab.edu
CONTRACT NUMBER: EY03040 (NEI)
SOURCE: CURRENT OPINION IN OPHTHALMOLOGY, (2000 Dec) 11 (6) 437-42. Ref: 57
Journal code: BB4. ISSN: 1040-8738.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: T
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010125

AB Vogt-Koyanagi-Harada (VKH) disease affects primarily persons who are Asian, Latino, Native American, or Asian Indian. Women appear to be affected more commonly than men, and VKH disease may occur at all ages, including childhood. Experimental data continue to support an **autoimmune** etiology for VKH **disease**, directed most probably against an **antigenic component** of the melanocyte, possibly tyrosinase or a tyrosinase-related protein. The clinical diagnosis of VKH disease continues to be based on physical findings. Newer imaging modalities such as magnetic resonance imaging, indocyanine green angiography, and digital image analysis have not added appreciably to our understanding of the condition. First-line **therapy** consists of high-dose corticosteroids, with use of corticosteroid-sparing agents for resistant or recalcitrant disease. Complications are the main cause of reversible and irreversible vision loss, with subretinal fibrosis and choroidal neovascular membranes having particularly poor prognoses.

L5 ANSWER 4 OF 26 MEDLINE
ACCESSION NUMBER: 1999221903 MEDLINE
DOCUMENT NUMBER: 99221903 PubMed ID: 10203740
TITLE: Q fever vaccine on trial for type I **diabetes**
.
AUTHOR: Bonn D
SOURCE: MOLECULAR MEDICINE TODAY, (1999 Apr) 5 (4) 143.
Journal code: CMK; 9508560. ISSN: 1357-4310.
PUB. COUNTRY: ENGLAND: United Kingdom
News Announcement
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19990913
Last Updated on STN: 19990913
Entered Medline: 19990902

L5 ANSWER 5 OF 26 MEDLINE
ACCESSION NUMBER: 95374257 MEDLINE
DOCUMENT NUMBER: 95374257 PubMed ID: 7646271
TITLE: [**Coxiella burnetii** endocarditis on a mechanical valvular prosthesis. Apropos of 2 cases].
Endocardite a **Coxiella burnetii** sur prothese valvulaire mecanique. A propos de deux cas.
AUTHOR: Stchepinsky O; Papo T; Amoyal P; Huisman J P; Theodose Y; Gaultier Y; Alexandre L; Piette J C
CORPORATE SOURCE: Centre William Harvey, Le Haut Boscq, Saint-Martin-d'Aubigny.
SOURCE: ARCHIVES DES MALADIES DU COEUR ET DES VAISSEAUX,

(1995 Apr) 88 (4) 511-5.
Journal code: 7SM; 0406011. ISSN: 0003-9683.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950930
Last Updated on STN: 19950930
Entered Medline: 19950921

AB The authors report two cases of prosthetic valve endocarditis due to **Coxiella burnetii**. The histories were chronic and complex suggesting an **auto-immune disease**: prolonged recurrent fever despite antibiotic **therapy** with a biological inflammatory syndrome whilst blood cultures remained negative. The first patient presented with prosthetic valve dehiscence and acute glomerulonephritis. The second patient had coagulation defects with prosthetic valve thrombosis, mesenteric adenopathy and congestive cardiac failure without prosthetic valve dysfunction. In suspected endocarditis with negative blood cultures, serological tests should be extended to intracellular pathogens difficult to identify and justifying specific and prolonged bactericidal **therapy** (fluoroquinolones, cyclines, rifampincine). Long-term serological surveillance is essential even when the outcome could have led to the termination of antibiotic **therapy**. Usually, antibiotic **therapy** provides a bacteriological cure, but **treatment** has to be continued for at least 3 years, and, in some patients, all their lives. Valve replacement is reserved for haemodynamic complications of the pathology which determine the ultimate prognosis.

L5 ANSWER 6 OF 26 MEDLINE
ACCESSION NUMBER: 95259084 MEDLINE
DOCUMENT NUMBER: 95259084 PubMed ID: 2485300
TITLE: [Experience in vaccinating farm animals for preventing Q fever in humans].
Opyt vaktsinatsii sel'skokhoziaistvennykh zhivotnykh kak mera profilaktiki likhoradki ku u liudei.
AUTHOR: Lisak V
SOURCE: TRUDY INSTITUTA IMENI PASTERA, (1989) 66 143-53, 174.
Journal code: VZG; 7709028. ISSN: 0202-1447.
PUB. COUNTRY: RUSSIA: Russian Federation
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 19950615
Last Updated on STN: 19970203
Entered Medline: 19950607

AB Vaccination of animals is indicated for cattle and sheep to prevent human infections deriving from these animals and inhibit the infertility in livestock. Both phase I and phase II are available. Vaccination is successful only when animals had been vaccinated as noninfected young, i. e. calves or lambs. Corpuscular vaccines are prevalent in immunoprophylaxis of domestic animals against **coxiellosis**. Such vaccines consist of formalin--inactivated, purified corpuscules **Coxiella burnetii** in phase

I or phase II suspended in isotonic saline. But only complete composition of particles **Coxiella burnetii** secures their immunogenicity. We assume that any vaccine prepared from phase II **Coxiella burnetii** can not be equivalent to a vaccine prepared from phase I **Coxiella burnetii**. We tried to ascertain the optimal dose of Q-fever vaccine **Coxiella burnetii**, strain Nine Mile phase I, for the immunization of cattle. The vaccine is called BODIBION and it is commercially produced by BIOVETA in Nitra, Czechoslovakia. It is recommended to vaccinate by **Coxiella** --free herds of cattle the heifers from the age of 3 months with a dose of 200 micrograms of vaccine with the possibility either to revaccinate with the same dose, i. e. 200 micrograms 3-4 weeks after the first dose, or to vaccinate with only a single dose, i. e. 200 micrograms, but with the addition of adjuvans. In Q-fever infested herds it is recommended to vaccinate with 500 micrograms of vaccine with the possibility of either to revaccinate after 3-4 weeks with a lower dose, i. e. 200 micrograms of vaccine, or to vaccinate with a single dose of 500 micrograms vaccine with the addition of adjuvans. The administration of adjuvans in the vaccination process seems to be perspective in the development of vaccines against **coxiellosis** in domestic animals. Its involvement may lower the recently used vaccination dose and may avoid the revaccination.

L5 ANSWER 7 OF 26 MEDLINE
ACCESSION NUMBER: 94221962 MEDLINE
DOCUMENT NUMBER: 94221962 PubMed ID: 7513276
TITLE: Chronic hepatitis C.
AUTHOR: Sherlock D S
CORPORATE SOURCE: Department of Medicine, Royal Free Hospital School of Medicine, University of London, United Kingdom.
SOURCE: DISEASE-A-MONTH, (1994 Mar) 40 (3) 117-96. Ref: 312
Journal code: EAV; 0370657. ISSN: 0011-5029.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940613
Last Updated on STN: 19960129
Entered Medline: 19940527

AB Formerly the diagnosis of acute and chronic non-A, non-B hepatitis was made by the exclusion of other causes. However, in 1989 cloning of an **antigenic component** of the hepatitis C virus (HCV) was reported. This led to first- and second-generation tests for antibody to HCV (anti-HCV) in serum. HCV has been associated with acute and chronic posttransfusion and sporadic non-A, non-B hepatitis, and with hepatocellular carcinoma. Viral HCV RNA can be estimated with the polymerase chain reaction test, but this technically difficult test is not generally available. The entire viral genome has been sequenced. The envelope region shows considerable variation, and mutant HCV infections are being described already. There are geographic variations in the prevalence of anti-HCV, but usually about 0.5% to 1% of healthy blood donors test positive. Parenteral exposure to blood, especially by transfusion or drug abuse, remains a certain means of acquiring HCV

infection. The method by which millions without parenteral risk factors acquire HCV remains uncertain. Vertical transmission and sexual and family spread occur only rarely. Body secretions are free of the virus. The mode of transmission may become clarified when tests for viral HCV as opposed to anti-HCV become generally available. Acute HCV infection usually is mild, and the chronic disease is also indolent. Carriers of hepatitis B virus or alcoholics who also test positive for HCV have more serious disease. Chronic HCV infection must be distinguished from autoimmune chronic active hepatitis. The most important difference is the response to corticosteroid **therapy**, which is good in autoimmune hepatitis and poor in HCV-related disease. Hepatocellular carcinoma can complicate HCV-related cirrhosis, usually about 20 years after infection with HCV. Recombinant interferon-alpha is used to **treat** chronic HCV disease, but selection of patients, dose, and duration of **therapy** are uncertain. In general, 50% of patients respond to the **treatment**, but 50% of these will have a relapse, with an overall response rate of 25%. Liver transplantation in patients with end-stage HCV disease usually is followed by infection of the graft.

L5 ANSWER 8 OF 26 MEDLINE
ACCESSION NUMBER: 92328461 MEDLINE
DOCUMENT NUMBER: 92328461 PubMed ID: 1626897
TITLE: Vaccines against **coxiellosis** and Q fever. Development of a chloroform:methanol residue subunit of phase I **Coxiella** burnetti for the immunization of animals.
AUTHOR: Williams J C; Peacock M G; Waag D M; Kent G; England M J; Nelson G; Stephenson E H
CORPORATE SOURCE: Office of the Scientific Director, National Institutes of Allergy and Infectious Diseases, Bethesda, Maryland 20982.
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1992 Jun 16) 653 88-111. Ref: 41
Journal code: 5NM; 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920821
Last Updated on STN: 19920821
Entered Medline: 19920813

AB We have demonstrated the safety, immunogenicity, and efficacy of the WC and CMR vaccines in guinea pigs. Vaccination of guinea pigs with either WC or CMR protects animals against challenge with virulent C. **burnetii**. A total of 2 micrograms of either WC or CMR vaccine was a significant priming dose. A total of 20 micrograms gave complete protection against lethal challenge. Detection of antibodies to phase II cells by microaglutination, after vaccination with either WC or CMR and before lethal challenge, correlated with the ability of guinea pigs to mount a protective immune response. The PD50 values for WC and CMR vaccines, administered as a single dose, were 0.3 and 1.4 micrograms per animal, respectively. In contrast, the PD50 values for the WC and CMR vaccines, administered

as two doses, were 0.83 and 0.72 micrograms per animal, respectively. Although the PD50 values for the two vaccines are similar, the CMR vaccine is preferred over the WC vaccine because it induces significantly fewer adverse reactions, and repeat injections can be given. Unvaccinated guinea pigs do not clear infectious microorganisms after challenge infection. Vaccination before challenge infection reduces the infectious load of C. burnetti in the blood and in various organs of the animals. When vaccinated animals were challenge infected and **treated** with rifampicin, the microorganisms were not eliminated from various organs. However, the combination of vaccination, challenge, and rifampicin **treatment** is effective in reducing the number of infectious microorganisms in some of these sites. We have demonstrated the safety and immunogenicity of the CMR vaccine in sheep and goats. Animals that were seropositive for one or more antigens developed significant levels of antibodies to alternate antigens, but no adverse reactions were observed at the site of s.c. injection of the CMR vaccine. This demonstrates that seropositive animals can be successfully immunized with this vaccine. These results also indicate that a long-term vaccination program using the CMR vaccine has the potential for producing animals with significant antibody titers to C. **burnetii** and perhaps lifelong immunity. The goal of a Q fever vaccination program is to produce immunized animals that are able to clear completely the infectious microorganisms. The appropriate vaccination schedule to render adult animals and their offspring "Q fever-free" should now be thoroughly investigated.

L5 ANSWER 9 OF 26 MEDLINE
ACCESSION NUMBER: 92111118 MEDLINE
DOCUMENT NUMBER: 92111118 PubMed ID: 1730186
TITLE: Pneumonia. Patient profiles, choice of empiric **therapy**, and the place of third-generation cephalosporins.
AUTHOR: Leedom J M
CORPORATE SOURCE: University of Southern California Medical Center, Los Angeles 90033.
SOURCE: DIAGNOSTIC MICROBIOLOGY AND INFECTIOUS DISEASE, (1992 Jan) 15 (1) 57-65. Ref: 30
Journal code: DMI; 8305899. ISSN: 0732-8893.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920308
Last Updated on STN: 19920308
Entered Medline: 19920219

AB Choosing appropriate antimicrobial **therapy** for patients with pneumonia requires knowledge of the etiologic agents seen in specific kinds of patients at specific times and places. For community-acquired pneumonia, there is an important difference in the agents seen in the normal and the compromised host. The normal host most often presents with viral, mycoplasmal, or pneumococcal pneumonia. The exact place of Chlamydia pneumoniae is still under study. A normal host who aspirates is at risk of anaerobic

pneumonia. Normal hosts with influenza may acquire superinfection with Streptococcus pneumoniae, Haemophilus influenzae, or Staphylococcus aureus. Under specific epidemiologic conditions, community-acquired pneumonia may be due to Legionella species, Yersinia pestis, Francisella tularensis, **Coxiella burnetii**, Chlamydia psittaci, a mycotic agent, or tuberculosis. Patients with chronic bronchitis and emphysema are predisposed to H. influenzae, Moraxella catarrhalis, and S. pneumoniae infections. HIV-infected patients are likely to have Pneumocystis carinii pneumonia and pneumonia due to cytomegalovirus, S. pneumoniae, and H. influenzae. Patients with **diabetes**, nursing-home patients, hospitalized patients, immuno-compromised patients, and patients with recent antibiotic **therapy** are predisposed to pneumonia due to Gram-negative aerobic bacilli of enteric and environmental origin. Initial **therapy** should be directed at the likely organism or organisms based on hospital susceptibility surveillance. In the normal host with community-acquired pneumonia, the **therapy** will often be penicillin G or erythromycin. In the patient predisposed to Gram-negative pneumonia, a third-generation cephalosporin with or without an aminoglycoside is the usual choice.

L5 ANSWER 10 OF 26 MEDLINE
ACCESSION NUMBER: 84265178 MEDLINE
DOCUMENT NUMBER: 84265178 PubMed ID: 6205020
TITLE: Myasthenia gravis. Identification of skeletal and heart muscle antigens not related to the acetylcholine receptor.
AUTHOR: Mehl V S; Lang R W
CONTRACT NUMBER: NS-08854-06 (NINDS)
SOURCE: JOURNAL OF NEUROIMMUNOLOGY, (1984 Aug) 6 (5) 347-60. Journal code: HSO; 8109498. ISSN: 0165-5728.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198409
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19840913

AB Myasthenia gravis (MG) is a neuromuscular **disease** thought to have an **autoimmune** etiology. The acetylcholine receptor has been considered the primary site of antibody binding; however, other muscle components may be involved in the pathogenesis of myasthenia gravis. This study describes a hypertonic sucrose extract of skeletal muscle (Muscle-HSE) that reacts with antibodies in myasthenic sera. The active component in Muscle-HSE is not the acetylcholine receptor as demonstrated by the inability of this extract to bind [125I]alpha-bungarotoxin. Muscle-HSE does, however, contain two distinct **antigenic components** reactive with MG sera. One antigen reacted with 70% (14/20) of myasthenic sera in the passive hemagglutination assay. This antigen was detected in the HSE of both skeletal muscle and heart, and was unaffected by **treatment** with Triton X-100. The second antigen reacted with 10% (2/20) of MG sera in the complement fixation assay, was unique to skeletal muscle, and was inactivated by Triton X-100.

L5 ANSWER 11 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1989:95171 BIOSIS
DOCUMENT NUMBER: BA87:49307
TITLE: MICROBIAL ECZEMA BACTERIAL FINDINGS SKIN TESTS STATE OF NATURAL RESISTANCE AND OF HUMORAL IMMUNITY.
AUTHOR(S): VIKTORINOVA M; KOUKALOVA D; MATOUSKOVA I; POLCROVA A; WERKMANNOVA A
CORPORATE SOURCE: RES. LAB. PHYSIOL. SKIN, I. P. PAVLOVA 6, 775 20 OLOMOUC, CZECH.
SOURCE: ACTA UNIV PALACKI OLOMUC FAC MED, (1988) 119 (0), 275-298.
CODEN: AUPMAF. ISSN: 0301-2514.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The paper confronts knowledge on etiopathogenesis of microbial eczema with the findings obtained by authors investigation. A detailed analysis of anamnestic, clinical, biochemical and immunological investigations, microbiological findings, epicutaneous skin tests and skin tests with microbial antigenic complex of staphylococci and pyogenic streptococci was performed in a series of patients with microbial eczema. The results obtained from case histories, clinical and microbiological examinations support the important role of bacterial infection in etiopathogenesis of the disease. Microbial eczema develops as a secondary disease in individuals predisposed to bacterial infection or contact allergy. Even after climinatiing a contact allergen the eczema is not cured - the original contact dermatitis develops into microbial eczema. Discrepancy in the results of skin tests with pyogenic bacterias reported in the literature where the importance of hypersensitivity is often obscured may be due to the fact that only some antigens are tested from the large scale of **antigenic components** of pyogenic cocci. By means of microbial antigenic complex which comprises besides bacterial cells numerous bacterial metabolites, hypersensitivity of immediate type was proved to Staphylococcus aureus in all patients under study and to Streptococcus pyogenes in 72% of patients with microbial eczema. Although microbial eczema belongs to allergic reactions of delayed, i.e. cell-mediated type of the allergy, allergy, of immediate type to pyogenic cocci appears to be also important in pathogenesis of the disease. The delayed type of reaction to microbial antigens may involve immune inflammatory responses, responses leading to damage of the organism, or may only indicate a previous bacterial infection (anamnestic reactions). No pathological biochemical and immunological finding demonstrated in patients with microbial eczema is typical of the disease and cannot serve as a diagnostic indicator. All found abnormalities can be explained by natural resistance and antibody response of the organism to a long-term or relapsing presence of pyogenic bacterias in eczematous foci. Local occurrence of pyogenic streptococci is not responsible for the origin of pyoderma but leads to the intensified activity of immune mechanisms, such as increased levels of lysozymes and IgG immunoglobulins in the serum, increased ASLO titers, high incidence of circulating immune complexes and increased phagocytic activity of leukocytes. Bacterial and immunological findings obtained from patients with microbial eczema may indicate the appropriate **treatment** category. With regard to a possible origin of **autoimmune diseases** (acute glomerulonephritis, rheumatic fever), antibiotics should be applied even in local

finding of pyogenic streptococci. On the contrary, bacterial finding of pyogenic staphylococci does not require the application of wide-spectrum antibiotics which may inhibit the favourable development of natural resistance and antibody formation of the organisms due to their immunosuppressive effects. This is manifested in clinical picture by recurrrences of the eczema or by the development of secondary pyoderma.

L5 ANSWER 12 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1986:245248 BIOSIS
DOCUMENT NUMBER: BA82:9752
TITLE: PRODUCTION OF HUMAN MONOCLONAL ANTIBODIES AGAINST EPSTEIN-BARR VIRUS-SPECIFIC ANTIGENS BY THE VIRUS-IMMORTALIZED LYMPHOBLASTOID CELL LINES.
AUTHOR(S): KOIZUMI S; FUJIWARA S; KIKUTA H; OKANO M; IMAI S; MIZUNO F; OSATO T
CORPORATE SOURCE: DEP. VIROLOGY, CANCER INST., HOKKAIDO UNIV. SCH. MED., SAPPORO 060, JPN.
SOURCE: VIROLOGY, (1986) 150 (1), 161-169.
CODEN: VIRLAX. ISSN: 0042-6822.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The possible production of human monoclonal antibodies against Epstein-Barr virus (EBV) was assessed through the EBV immortalization technique. When individual lymphocyte samples from 50 clinical patients and healthy donors were immortalized by EBV, 4 lymphoblastoid lines yielded antibodies to EBV antigens. These positive lines were cloned and each line yielded cultures that secreted monoclonal antibodies against either viral capsid antigen (VCA) or membrane antigen (MA) component. Above all, a clonal line TAKA-SP-8 produced 5 .mu.g MA antibody/106 cells/ml for more than 12 months. The culture fluid specifically immunoprecipitated a single polypeptide with a size of 93K from both P3HR-1 and B95-8 cell extracts. FUKA-SP-3, on the other hand, secreted 5 .mu.g VCA antibody/106 cells/ml for at least 8 months. This antibody recognized two polypeptides with sizes of 123K and 120K, from P3HR-1 and B95-8 cell extracts, respectively. When B95-8 and P3HR-1 EBV were **treated** with the human MA monoclonal, both nuclear antigen (EBNA) synthesis and early antigen (EA) induction were strongly inhibited. All EBV antibody-producing cultures were exclusively achieved from splenic lymphocytes of patients with **autoimmune diseases**, but not from other donors.

L5 ANSWER 13 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-656919 [75] WPIDS
DOC. NO. CPI: C2001-193275
TITLE: Polynucleotide construct encoding a processing component derived from N-terminal region of Hepatitis Virus ORF2 and an antigenic polypeptide useful for enhancing immune response to the polypeptide in an animal.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): ANDERSON, D A; LI, F; PURCELL, D F J
PATENT ASSIGNEE(S): (MACF-N) MACFARLANE BURNET CENT MEDICAL
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

WO 2001073078 A1 20011004 (200175)* EN 47
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC
MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ
DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE
KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO
NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ
VN YU ZA ZW
AU 2001043941 A 20011008 (200208)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001073078 A1		WO 2001-AU353	20010330
AU 2001043941 A		AU 2001-43941	20010330

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001043941 A	Based on	WO 200173078

PRIORITY APPLN. INFO: AU 2000-6616 20000331
AN 2001-656919 [75] WPIDS
AB WO 200173078 A UPAB: 20011220
NOVELTY - An isolated polynucleotide construct (I) comprising a sequence encoding fusion protein having a processing component (II) comprising sequence of 36 or 50 amino acids as given in the specification; and an **antigenic component** (III), where (II) provides heterogenous processing of (III) when (I) is expressed in a host cell and results in enhancement of immune response to (III), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (IV) encoding (II);

(2) an isolated cell (V) transfected with (I) or (IV); and

(3) nucleic acid vaccine comprising (I) or (IV).

ACTIVITY - Cytostatic; Immunosuppressive; Antiparasitic; Antibacterial; Virucide.

MECHANISM OF ACTION - Vaccine (claimed).

Balb/C mice were inoculated with plasmid constructs or nucleic acid vaccines encoding N-terminal fusion proteins of sig1 (sig1-2.1), sig2 (sig2-2.1) or sig3 (sig3-2.1) with glutathione S-transferase (GST) and desired antigens, by standard methods such as gene gun or intramuscular injection, and the immune response in animals receiving each vaccine was compared by methods such as specific antibody isotype profile, T-cell proliferative responses, and cytolytic T-cell responses. Results showed that the sig1, sig2 and sig3 and related peptides derived from HEV PORF2 modulated and enhanced the immune response to fusion protein antigens by virtue of heterogeneous patterns of intracellular processing and localization.

USE - (I) is useful for enhancing and immune response to an desired antigenic polypeptide especially viral capsid polypeptide in an animal (claimed). (I) is also useful in manufacture of a medicament for the **treatment** or prophylaxis of conditions or infections including cancer or pre-cancerous conditions,

autoimmune diseases, viral, bacterial or parasitic infections in animals including humans and other mammals such as fish or birds.
Dwg.0/7

L5 ANSWER 14 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-514530 [56] WPIDS
DOC. NO. CPI: C2001-153759
TITLE: New vaccine composition comprising a denatured tissue from a pathogen-infected host, useful for **treating** or preventing e.g. pathogen-induced infections, tumors, immune disorders, cancers.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): JIRA, V; JIRATHITICAL, V
PATENT ASSIGNEE(S): (JIRA-I) JIRA V; (JIRA-I) JIRATHITICAL V
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001054717	A1	20010802	(200156)*	EN	56

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001034616 A 20010807 (200174)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001054717	A1	WO 2001-US2811	20010129
AU 2001034616	A	AU 2001-34616	20010129

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001034616	A Based on	WO 200154717

PRIORITY APPLN. INFO: US 2000-227520P 20000824; US 2000-494607 20000131
AN 2001-514530 [56] WPIDS
AB WO 200154717 A UPAB: 20011001
NOVELTY - A composition (I) for **treating** or preventing a pathogen-induced infection in a host comprising a denatured tissue from a pathogen-infected host, is new.
DETAILED DESCRIPTION - A composition (I) for **treating** or preventing a pathogen-induced infection in a host comprising a denatured tissue from a pathogen-infected host, may also comprise a denatured antigen of an infection-inducing pathogen.
INDEPENDENT CLAIMS are also included for the following:
(1) methods of **treating** or preventing a pathogen-induced infection in an animal by contacting a mucosal

surface of an animal with (I), or administering a composition comprising a denatured pathogen-containing tissue derived from another animal infected with the pathogen;

(2) a process for producing a pharmaceutical composition for **treating** or preventing a pathogen infection, tumor or immune disorder, by reducing a tissue-derived from a pathogen-infected animal, tumor or organ affected by the immune disorder;

(3) **treating** a subject with an **autoimmune disorder** or inflammatory reaction comprises administering a composition containing a compound comprising a denatured tissue of the subject rejected by an autoimmune or inflammation reaction;

(4) a vaccine (II) for protecting a host against a virus and for transmucosal administration comprising a denatured tissue or a cell infected with the virus, a denatured virus or viral antigen, or its fragment;

(5) a method for protecting a host from a viral infection by administering (II) to the host;

(6) a method of decreasing frequency of virus transmission or for **treating** virus infection by administering a composition comprising a denatured virus-infected cell or denatured virus;

(7) oral vaccines (III) comprising a denatured antigen derived from a pathogen-infected host tissue, and which elicits a mucosal immune response in a subject, or comprising at least one denatured cancer antigen derived from a cancer tissue or cell, for cancer **treatment**;

(8) a method for inducing a systemic response in a subject by orally administering (III); and

(9) a vaccine comprising a heat-denatured antigen derived either from a pathogen, pathogen-infected tissue, a malignant tissue, or a benign tissue, which is subject to an immune reaction.

ACTIVITY - Cytostatic; immunosuppressive; immunomodulator; antimicrobial; antiviral; antifungal; anti-inflammatory. No biodata is given.

MECHANISM OF ACTION - Vaccine.

USE - The vaccine is useful for **therapy** and prophylaxis of pathogen-induced infections, tumors, and immune disorders. The composition can be used against HIV, to induce immunoprotective factors protective against progression of infection, it can also possess immunomodulatory, antimicrobial, antiviral, antifungal, anti-inflammatory, antitumor or anti-cancer activity.

Dwg.0/2

L5 ANSWER 15 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-257888 [26] WPIDS
DOC. NO. CPI: C2001-077728
TITLE: Use of effectors of GTPase as target in a in vitro/vivo assay for detecting substances for prophylaxis, **treatment** of cancer, cell migration disorders, e.g. Alzheimer's, infectious diseases, **diabetes**, atherosclerosis.
DERWENT CLASS: B04 D16
INVENTOR(S): CHRISTOPHORIDIS, S; MURPHY, C; NIELSEN, E; ZERIAL, M; DE RENZIS, S
PATENT ASSIGNEE(S): (PLAC) MAX PLANCK GES FOERDERUNG WISSENSCHAFTEN
COUNTRY COUNT: 28
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001020022	A1	20010322	(200126)*	EN	76
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: CA JP US					
EP 1088898	A1	20010404	(200126)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001020022	A1	WO 2000-EP9130	20000918
EP 1088898	A1	EP 1999-118385	19990916

PRIORITY APPLN. INFO: EP 1999-118385 19990916

AN 2001-257888 [26] WPIDS

AB WO 200120022 A UPAB: 20010515

NOVELTY - Use of an effector of a GTPase as a target in an in vitro or in vivo assay to detect substances useful as pharmaceutical agents for the prophylaxis and/or **treatment** of cancer and other proliferative, invasive or cell migration disorders, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a kit for carrying out the assay, comprising a GTPase effector/regulator molecule labeled by a covalent modification or by radioactivity suitable for use in the assay.

ACTIVITY - Anti-HIV; Tuberculostatic; Protozoacide; Antidiabetic; Nootropic; Neuroprotective; Dermatological; Antipsoriatic; Antiinflammatory; Antiallergic; Antipyretic; Antibacterial; Cytostatic; Gynecological; Antiatherosclerotic. No supporting data is given.

MECHANISM OF ACTION - Gene **therapy**; Inhibitor of cancer cell growth; Stimulator of endocytic transport and phagosome maturation in cells infected by intracellular parasites.

USE - The method is useful for detecting substances useful as pharmaceutical agents for the prophylaxis or **treatment** of cancer and other proliferative, invasive or cell migration disorders such as endometriosis, atherosclerosis, inflammatory and allergic diseases, infectious diseases, **diabetes**, Alzheimer's disease and skin repair diseases such as psoriasis. The infectious diseases include AIDS, tuberculosis, pseudotuberculosis, cholera, malaria, gastroenteritis, enteric fever, typhus, diseases caused by pathogens such as Mycobacterium, Staphylococcus, Toxoplasma, Trypanosoma, Listeria, Salmonella, Legionella, Leishmania, **Coxiella**, Shigella, Yersinia, Neisseria, Vibrio, Bartonella, or any other infectious diseases caused by any infectious agent that infects cells by the endocytic route and resides intracellularly in phagosomes escaping the cellular killing mechanisms. The cancer includes benign tumor, malignant tumor, carcinoma, leukemia, glioma or a neuroblastoma, in particular lung carcinoma, osteosarcoma, lymphoma, breast, bile, intestine, kidney, ovary, stomach, brain, prostate, liver and every tumor that invades other tissues and organs distinct from its site of origin (claimed).

ADVANTAGE - The assay is highly sensitive and advantageous in the selectivity of the targets.

Dwg.0/0

L5 ANSWER 16 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-558255 [51] WPIDS
DOC. NO. NON-CPI: N2000-413119
DOC. NO. CPI: C2000-166234
TITLE: Use of CD38 or fragments that inhibit binding to a non-follicular or follicular dendritic cell in the manufacture of an adjuvant for use in immunotherapy.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): MACPHERSON, G G; WYKES, M N
PATENT ASSIGNEE(S): (ISIS-N) ISIS INNOVATION LTD
COUNTRY COUNT: 91
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000048631	A2	20000824	(200051)*	EN	42
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000025623	A	20000904	(200103)		
EP 1152771	A2	20011114	(200175)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000048631	A2	WO 2000-GB559	20000217
AU 2000025623	A	AU 2000-25623	20000217
EP 1152771	A2	EP 2000-903870	20000217
		WO 2000-GB559	20000217

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000025623	A	Based on	WO 200048631
EP 1152771	A2	Based on	WO 200048631

PRIORITY APPLN. INFO: GB 1999-3664 19990217
AN 2000-558255 [51] WPIDS
AB WO 200048631 A UPAB: 20001016
NOVELTY - Use of at least a portion or analogue of CD38 (I) which can inhibit the binding to a non-follicular dendritic cell (DC) or a follicular dendritic cell (FDC) and which can stimulate a DC or FDC, for use in the manufacture of an adjuvant for use in immunotherapy, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) causing maturation of DCs comprising contacting DCs ex vivo with CD38 or a portion or analogue as in (I);

(2) a vaccine comprising an antigen and an adjuvant, where the adjuvant comprises CD38 or a portion or analogue as in (I);
(3) an ex vivo mature DC that has been made using the method of (1);
(4) a vaccine comprising the DC of (3);
(5) stimulating T cells specific to an epitope in vitro comprising contacting the T cells with the DC of (3)1;
(6) a protein comprising:
(i) the native ligand present on a FDC or DC which binds CD38 and is free of FDC or DC cell membrane, excluding CD31;
(ii) a protein which is at least 70% homologous to (i) and binds CD38; or
(iii) a fragment of (i) or (ii) which retains the ability to bind CD38;
(7) a polynucleotide that encodes the peptide of (6.i), (6.ii), or (6.iii);
(8) an antibody that binds the peptide of (6.i), (6.ii), or (6.iii) to inhibit the binding of CD38;
(9) identifying an adjuvant comprising determining whether a substance competes with CD38 for the CD38 ligand present on FDCs or DCs, or the peptide of (6.i), (6.ii), or (6.iii); and
(10) an inhibitor of a CD38 ligand which specifically inhibits the activation of the ligand by CD38.
ACTIVITY - Immunostimulant; immunosuppressant.
No biological data given.
MECHANISM OF ACTION - None given.
USE - CD38, or portion or analogue of it can be used in the manufacture of an adjuvant for use in immunotherapy. They can also be used as an adjuvant in a vaccine. The ex vivo mature DC can be used in a method of **treating** a human or animal body. The DC can also be used in stimulating a T cell response in vivo, and can also be used in a vaccine. Adjuvants identified using the method of (9) can be used in vaccines and for the manufacture of an adjuvant for immunotherapy. The inhibitors can be used in immunosuppression (all claimed). The inhibitor may be used to **treat diseases**, particularly **autoimmune diseases**, or immune responses caused by a foreign agent. The antibody can be used in a method for identifying adjuvants.
ADVANTAGE - None given.
Dwg.0/6

L5 ANSWER 17 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-339492 [29] WPIDS
DOC. NO. NON-CPI: N2000-254915
DOC. NO. CPI: C2000-102962
TITLE: New artificial antigen presenting cells useful for isolating and expanding T cells, and modulating T cell responses for the **treatment** of e.g. **autoimmune diseases**, allergies.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): ALBANI, S
PATENT ASSIGNEE(S): (ALBA-I) ALBANI S
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000023053	A2	20000427	(200029)*	EN	179

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC
MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM
EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD
SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000011293 A 20000508 (200037)
EP 1123086 A2 20010816 (200147) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK
NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000023053	A2	WO 1999-US24666	19991019
AU 2000011293	A	AU 2000-11293	19991019
EP 1123086	A2	EP 1999-955116	19991019
		WO 1999-US24666	19991019

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000011293	A	Based on	WO 200023053
EP 1123086	A2	Based on	WO 200023053

PRIORITY APPLN. INFO: US 1998-105018P 19981020
AN 2000-339492 [29] WPIDS
AB WO 200023053 A UPAB: 20000617
NOVELTY - Artificial antigen presenting cells (APC) comprising combinations of MHC:antigen complex with accessory molecules, co-stimulatory molecules, adhesion molecules, cell modulation molecule, irrelevant molecule, cholesterol, or solid support components, are new.

DETAILED DESCRIPTION - Artificial APCs comprising liposome, MHC, antigen, and accessory molecule components in combination with at least one of the following components: co-stimulatory molecule, cell modulation molecule, adhesion molecule, irrelevant molecule, cholesterol, or solid support components, the **antigen component** is in contact with at least the MHC component, the MHC and accessory components are in contact with at least one of the components, and the accessory molecule components provide for a stabilizing property to an interaction between a T cell receptor and MHC and antigen compounds.

INDEPENDENT CLAIMS are also included for the following:

(1) a method of making an artificial antigen presenting cell comprising:

(a) obtaining an MHC:antigen complex of interest;

(b) contacting the complex with a lipid and cholesterol, and forming a lipid membrane-associated the complex; and

(c) contacting the membrane-associated MHC:antigen complex with a molecule of interest selected from an accessory molecule, a co-stimulatory molecule, a cell modulation molecule, an adhesion molecule, an irrelevant molecule, cholesterol, GM-1 protein, cholera toxin beta subunit protein or a label;

(2) a method of identifying T cells specific for an antigen of interest comprising:

(a) obtaining a biological sample containing T cells specific for an antigen of interest;

(b) preparing an artificial APC, which contain the antigen;

(c) contacting the biological sample with the APC to form an artificial APC:T cell complex; where at least one element of the artificial antigen presenting cell is associated with a label, the element is selected from the antigen, an irrelevant molecule, a lipid layer, a lipid, an MHC molecule component, a co-stimulatory component, a cell modulation component, or an accessory molecule component; and

(d) detecting the label;

(3) a method of isolating T cells specific for an antigen of interest by employing the steps of (3a-c), removing the artificial APC:T cell complex from the biological sample; and separating T cells specific for the antigen from the artificial APC:T cell complex;

(4) a method of modulating T cell response by isolating T cells specific for an antigen of interest employing the method of (9); and contacting the isolated T cells with an artificial APC which has the antigen or its homologue, the artificial APC further having at least one molecule selected from an accessory molecule component, a co-stimulatory component, an adhesion component or a cell modulation component;

(5) methods of **treating** a condition in a subject which would be benefited by altering the functional pattern of cytokine production by certain antigen-specific T cells to increase or decrease Th-2 or Th-1 response comprising:

(a) isolating T cells specific for an antigen capable of triggering a Th-1 or Th-2 response upon recognition of the antigen by the subject's T cells; and

(b) combining the isolated T cells with an artificial APC having an MHC component capable of binding the antigen and a co-stimulatory molecule component comprising B7-2 or B7-1;

(6) a kit for isolation and/or modulation of T cells specific for an antigen of interest comprising artificial APCs, solid supports, reagents or an immunomodulatory column device;

(7) an immunomodulatory column comprising a multiple compartments having a channel interconnecting adjacent compartments, positioned in relation to one another in series, the channels having a means to isolate these compartments from one another, where the compartments further have at least one entrance and exit port for receiving or expelling, respectively, a flowable medium, the ports have a means to close to impede the flowable medium, and the compartments is optionally comprised of the components solid supports or artificial APCs.

ACTIVITY - Cytostatic; anti-sclerotic; anti-allergic; antiarthritic; antiviral; immunosuppressive.

MECHANISM OF ACTION - T cell response modulator.

USE - Artificial APCs may be used for isolating T cells specific for an antigen of interest, as well as for modulating and modifying T cell responses. These may also be used for the **treatment** of a condition in an individual who would be benefited by modulating the functional pattern of active factors expressed by a T cell. Conditions which may be improved by altering the functional pattern of response toward a Th2 response include e.g. type 1 **diabetes** mellitus, multiple sclerosis rheumatoid arthritis, juvenile rheumatoid arthritis dermatomyositis, and uveitis, cancer, viral or bacterial infection, an

autoimmune disease or an allergy (to dust, animal skin bypass products, vegetables, fruits, pollen or chemicals). The APCs are useful for manipulating the T cell responses by which **treatment** can be provided for numerous disease states.

ADVANTAGE - The present invention is more versatile compared with prior arts. It is not concerned with detecting natural APCs, instead directed to the isolation and manipulation of antigen-specific T cells. The use of co-stimulatory, adhesion and other accessory molecules in a free floating format helps to both anchor and direct the interaction between MHC:antigen:accessory molecule and T cell receptors by providing a means by which T cells in the sample will be represented with a structure more similar with that found in the natural state. The free floating MHC component is able to participate in the migration or concentration of complexes in capping which is important to improved binding and activation of bound t cells. Moreover, no cell proliferation is necessary to identify and isolate antigen-specific T cells. Addition of accessory molecules allows for substantially improved binding associated and manipulation of T cells important in the identification and stimulation of antigen-specific T cells.

Dwg.0/24

L5 ANSWER 18 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1999-315490 [27] WPIDS
DOC. NO. NON-CPI: N1999-235821
DOC. NO. CPI: C1999-093393
TITLE: Identifying Mycobacterium species is useful for detecting leprosy, tuberculosis, sarcidosis, Crohn's **disease** and other **autoimmune diseases**.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): GOERDAYAL, S; HOUTHOFF, H; KOLK, A; KROON-SWART, S; KUYPER, S; PEREIRA ARIAS-BOUDA, L; VAN DER MEULEN, R
PATENT ASSIGNEE(S): (KREA-N) KREATECH BIOTECHNOLOGY BV
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 921397	A1	19990609	(199927)*	EN	10
R: AL AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
WO 9930162	A1	19990617	(199931)	EN	
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9914462	A	19990628	(199946)		
EP 1038181	A1	20000927	(200048)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE					
CN 1284169	A	20010214	(200130)		
JP 2001526393	W	20011218	(200203)		26
MX 2000005608	A1	20010501	(200227)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 921397	A1	EP 1997-203851	19971208
WO 9930162	A1	WO 1998-NL701	19981208
AU 9914462	A	AU 1999-14462	19981208
EP 1038181	A1	EP 1998-958404	19981208
		WO 1998-NL701	19981208
CN 1284169	A	CN 1998-813534	19981208
JP 2001526393	W	WO 1998-NL701	19981208
		JP 2000-524669	19981208
MX 2000005608	A1	MX 2000-5608	20000607

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9914462	A	Based on	WO 9930162
EP 1038181	A1	Based on	WO 9930162
JP 2001526393	W	Based on	WO 9930162

PRIORITY APPLN. INFO: EP 1997-203851 19971208
AN 1999-315490 [27] WPIDS
AB EP 921397 A UPAB: 20011203
NOVELTY - The method for identifying a Mycobacterium species comprises, contacting at least one immuno-cross reactive **antigen component** of a mycobacterial species with a sample of body fluid (human/animal), contacting at least one antibody and detecting the presence of antigen-antibody complexes.

DETAILED DESCRIPTION - The antibody is capable of reacting with a mycobacterial antigen with the body fluid and the method further comprises identifying the Mycobacterium species present in the body fluid.

An INDEPENDENT CLAIM is also included for a diagnostic test kit comprising a support, on which at least immuno-cross reactive **antigen component** of a mycobacterial species and at least one antibody which is capable of reacting with a mycobacterial antigen and which does not react with the immuno-cross reactive **antigen component**, are bound and a means for detecting the presence of antigen-antibody complexes.

USE - The method is useful for identifying a Mycobacterium species responsible for a mycobacterial infection (claimed), responsible for a number of diseases e.g. leprosy, tuberculosis, sarcidosis, Crohn's **disease** and **autoimmune diseases** e.g. **autoimmune** dermatitis, rheumatoid arthritis and **diabetes**. The method is also useful for monitoring the different stages of a **treatment** of a mycobacterial disease and for testing whether an individual has been vaccinated for a mycobacterial disease.

ADVANTAGE - The performance of the test is successful and reliable and mycobacterial infections are sufficiently manifested in saliva. The collection and use of saliva is easy to collect, even under difficult field conditions (especially encountered in Third World countries), saliva is non-invasive, requires minimal training to collect and has reduced biohazard risk in collection, transport and testing especially in areas with a high incidence of HIV-infections. Also the antigen preparations provide reliable test results, are sufficiently stable to be stored for a prolonged period

of time without effecting the reliability of the diagnostic test.

L5 ANSWER 19 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1998-250936 [22] WPIDS
DOC. NO. NON-CPI: N1998-198138
DOC. NO. CPI: C1998-078175
TITLE: Use of compositions containing phospholipase A2 or antiserum to it - for **treating** e.g. neoplasms, inflammation, **auto-immune disease**, allergic **disease**, asthma, renal failure or parasitic or bacterial infections.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): SHANAHAN-PRENDERGAST, E; SHANAHANPREDERGAST, E
PATENT ASSIGNEE(S): (SHAN-I) SHANAHAN-PRENDERGAST E; (SHAN-I) SHANAHAN-PREDERGAST E
COUNTRY COUNT: 80
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9810776	A1	19980319	(199822)*	EN	34
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9741323	A	19980402	(199833)		
EP 1019068	A1	20000719	(200036)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
AU 741943	B	20011213	(200210)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9810776	A1	WO 1997-IB1091	19970910
AU 9741323	A	AU 1997-41323	19970910
EP 1019068	A1	EP 1997-939108	19970910
		WO 1997-IB1091	19970910
AU 741943	B	AU 1997-41323	19970910

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9741323	A	Based on	WO 9810776
EP 1019068	A1	Based on	WO 9810776
AU 741943	B	Previous Publ.	AU 9741323
		Based on	WO 9810776

PRIORITY APPLN. INFO: US 1996-25179P 19960911
AN 1998-250936 [22] WPIDS
AB WO 9810776 A UPAB: 19980604
The following are claimed: (A) **treatment** of neoplasm, parasitic and bacterial infections in a mammal by administering an agent comprising venom and/or mammalian, plant or insect anti-serum

reactive with at least 1 Phospholipase A2 (PLA2) enzyme; (B) a method of **treating** a mammal prophylactically to prevent neoplastic development by administering a vaccine containing venom and/or mammalian, plant or insect PLA2 enzymes or part or these as the principal **antigen component**; (C) a pharmaceutical formulation containing venom and/or mammalian plant or insect anti-serum to PLA2 enzyme or part of these in combination with anti-serum to phospholipase C (PLC) enzyme or part of it or inhibitory compounds to PLC for use as a **therapeutic** agent for the **therapy** of a neoplastic condition in a human or animal; (D) a pharmaceutical formulation containing at least 1 venom or venom component as antigen and/or mammalian, plant or insect PLA2 enzyme as antigen in combination with PLC enzyme; (E) a method of inoculation of human or animal with a combination of at least 2 PLA2 enzyme types; (F) a method of early detection of neoplastic disease by utilising the detection of enhanced PLA2 levels in patients, and (G) a method of targeting cancer cells by use of type I and/or type II PLA2 as targeting agent with a hydrophilic tail.

The inhibitory compounds to PLC may be e.g. EDTA, phenanthroline, chloromercuribenzoic acid, iodoacetic acid, or 1-oleoyl-2-acetyl-sn-glycerol (OAG). The anti-serum is reactive with at least 2 phospholipase A2 type enzymes, 1 of which is type I, II, III or IV. The anti-serum is either monoclonal or polyclonal.

USE - The methods can be used for **treating** neoplastic disease, contact dermatitis, asthma, psoriasis, bronchitis, rheumatoid arthritis, osteoarthritis, gout, rheumatic carditis, **autoimmune diseases**, allergic **diseases**, septic shock, renal failure, pancreatitis, myasthenia gravis and ocular and dermal inflammatory diseases, splenomegaly, metastatic spread of neoplasm, collagen vascular disease, myocardial ischaemia, cellular chemotaxis, depression, erythema, vascular permeability resultant from enhanced production of PGE2, acne, atopic diseases, allergic conjunctivitis, schizophrenia, Reiter's syndrome, Raynaud's phenomenon, lupus, Crohn's and Graves disease, or parasitic or bacterial infections e.g. malaria, leishmania, trypanosomia or toxoplasma (all claimed).

Dwg.0/7

L5 ANSWER 20 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1997-470646 [43] WPIDS
DOC. NO. CPI: C1997-149552
TITLE: Prevention or **treatment** of **auto -immune disease** using **Coxiella** or derived antigens - especially for insulin-dependent **diabetes**, also to improve survival of transplanted islet cells.
DERWENT CLASS: B04 D16
INVENTOR(S): COWDEN, W B; GAZDA, L S; LAFFERTY, K J
PATENT ASSIGNEE(S): (AUSU) UNIV AUSTRALIAN NAT; (COWD-I) COWDEN W B; (GAZD-I) GAZDA L S; (LAFF-I) LAFFERTY K J
COUNTRY COUNT: 78
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9733614	A1	19970918	(199743)*	EN	34

RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG

W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI
GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD
MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT
UA UG US UZ VN YU

AU 9719171 A 19971001 (199805)
ZA 9702232 A 19980128 (199810) 35
EP 886528 A1 19981230 (199905) EN
R: AL AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC NL PT
RO SE SI
JP 2000506172 W 20000523 (200033) 33
AU 720327 B 20000525 (200034)
NZ 331536 A 20000728 (200043)
NZ 503143 A 20010629 (200140)
US 2001051162 A1 20011213 (200204)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9733614	A1		WO 1997-AU161	19970314
AU 9719171	A		AU 1997-19171	19970314
ZA 9702232	A		ZA 1997-2232	19970314
EP 886528	A1		EP 1997-906937	19970314
			WO 1997-AU161	19970314
JP 2000506172	W		JP 1997-532124	19970314
			WO 1997-AU161	19970314
AU 720327	B		AU 1997-19171	19970314
NZ 331536	A		NZ 1997-331536	19970314
			WO 1997-AU161	19970314
NZ 503143	A		NZ 1997-503143	19970314
US 2001051162	A1	Cont of	WO 1997-AU161	19970314
		Cont of	US 1999-142597	19990305
			US 2001-887773	20010621

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9719171	A	Based on	WO 9733614
EP 886528	A1	Based on	WO 9733614
JP 2000506172	W	Based on	WO 9733614
AU 720327	B	Previous Publ.	AU 9719171
		Based on	WO 9733614
NZ 331536	A	Based on	WO 9733614
NZ 503143	A	Div in	NZ 512067

PRIORITY APPLN. INFO: AU 1996-8703 19960314
AN 1997-470646 [43] WPIDS
AB WO 9733614 A UPAB: 19971030
The effect of an **autoimmune disease** in a mammal is prevented, inhibited, delayed or otherwise alleviated by administration of a **Coxiella** species, or one of its antigens (or analogues or homologues) (I). Also claimed are: (i) a method for prolonging the survival of transplanted islet tissue comprising administration of (I); and (ii) a composition comprising (I).

USE - The method is used in humans or other animals, specifically to **treat** or prevent insulin-dependent

diabetes mellitus (but may also be effective in pernicious anaemia, chronic hepatitis, ulcerative colitis, primary biliary cirrhosis, multiple sclerosis and systemic lupus erythematosus) or a disease that affects survival of transplanted pancreatic islet tissue.

ADVANTAGE - (I) is sufficiently safe for general use (contrast complete Freund's adjuvant (FCA) or bacillus Calmette-Guerin) and is more effective at diverting the immune response away from destructive autoimmunity.

Dwg.1/2

L5 ANSWER 21 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1993-102961 [13] WPIDS
DOC. NO. NON-CPI: N1993-078260
DOC. NO. CPI: C1993-045398
TITLE: Recombinant DNA encoding human V-beta-17 chain of T-cell receptors - and corresp. murine constant region, used to raise antibodies to the variable chain for prevention and diagnosis of **auto -immune disorders** and septic conditions.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BESNARDEAU, L; MALISSEN, B; ROMAGNE, F; VAN, AGHTOVEN A; VAN, AGTHOVEN A
PATENT ASSIGNEE(S): (IMMU-N) IMMUNOTECH SA
COUNTRY COUNT: 19
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 534878	A1	19930331	(199313)*	FR	22
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE					
CA 2079197	A	19930327	(199323)	FR	
FR 2681875	A1	19930402	(199326)		36
JP 05276956	A	19931026	(199347)		13

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 534878	A1	EP 1992-430022	19920924
CA 2079197	A	CA 1992-2079197	19920925
FR 2681875	A1	FR 1991-12148	19910926
JP 05276956	A	JP 1992-258117	19920928

PRIORITY APPLN. INFO: FR 1991-12148 19910926
AN 1993-102961 [13] WPIDS
AB EP 534878 A UPAB: 19931112
New recombinant DNA (I) comprises a chimferic sequence, encoding the beta-chain of the T-cell antigen receptor (TCR), which consists of (1) a human sequence (Ia) encoding the whole of a variable part (Vbeta17) and its head, diversity (Dbeta) and junction (Jbeta) sequence, and (2) a murine sequence (Ib) encoding the complementary constant part (Cbeta1 or Cbeta2) absent from (Ia). (Ia) and (Ib) are joined at a natural or artificial restriction site.

Also new are antibodies (Ab) raised against cells which express (I).

The restriction site is naturally present in the human Cbeta sequence (esp. it is a BglII site) and it is artificially created (esp. by mutagenesis) in the murine sequence. Most pref. (Ia) includes a short portion (esp. about 60 nucleotides) of the human Cbeta region.

USE/ADVANTAGE - Ab are directed against the Vbeta17 chain of human TCR. They can distinguish Vbeta17 element in a human T-cell population so can be used e.g. to diagnose particular diseases or defects. Ab can also be used to **treat** or prevent **autoimmune diseases** (e.g. rheumatoid arthritis, **diabetes**, Shorgen's disease and lupus); infections (esp. those caused by mycobacteria and Staphylococci) or leukaemias where these involve proliferation of T cells carrying the Vbeta17 chain. The usual daily dose of Ab (coupled to ricin A) is 0.05-0.2 mg/kg. Ab can also be used to purify the **antigenic components** of TCR chain and for imaging T-cell distribution.

Dwg.0/6

ABEQ FR 2681875 A UPAB: 19931116

New recombinant DNA (I) comprises a chimferic sequence, encoding the beta-chain of the T-cell antigen receptor (TCR), which consists of (1) a human sequence (Ia) encoding the whole of a variable part (Vbeta17) and its head, diversity (Dbeta) and junction (Jbeta) sequence, and (2) a murine sequence (Ib) encoding the complementary constant part (Cbeta1 or Cbeta2) absent from (Ia). (Ia) and (Ib) are joined at a natural or artificial restiction site.

Also new are antibodies (Ab) raised against cells which express (I).

The restriction site is naturally present in the human Cbeta sequence (esp. it is a BgIII site) and it is artificially created (esp. by mutagenesis) in the murine sequence. Mos pref. (Ia) includes a short portion (esp. about 60 nucleotides) of the human Cbeta region.

USE/ADVANTAGE - Ab are directed against the Vbeta17 chain of human TCR. They can distinguish Vbeta17 element in a human T-cell population so can be used e.g. to diagnose particular diseases or defects. Ab can also be used to **treat** or prevent **autoimmune diseases** (e.g. rheumatoid arthritis, **diabetes**, Shorgen's disease and lupus); infections (esp. those caused by mycobacteria and Staphylococci) or leukaemias where these involve proliferation of T-cells carrying the Vbeta17 chain. The usual daily dose of Ab (coupled to ricin A) is 0.05-0.2 mg/kg. Ab can also be used to purify the **antigenic components** of TCR chain and for imaging T-cell distribution.

Dwg.0/6

ABEQ JP 05276956 A UPAB: 19940111

L5 ANSWER 22 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1991-117337 [16] WPIDS
DOC. NO. NON-CPI: N1991-090330
DOC. NO. CPI: C1991-050472
TITLE: Isolation of specific target antibody from physiological soln. - used to prepare pharmaceutical compsns. for **treating** allergy or **auto immune disease**.
DERWENT CLASS: B04 D16 P34 P41
INVENTOR(S): CHENOWETH, D; HARDWICK, A R; LAKE, W C; SMITH, A K; HARDWICK, R A; CHENOWETH, D E; HARDWICH, R A

PATENT ASSIGNEE(S): (BAXT) BAXTER INT INC
COUNTRY COUNT: 19
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9104059	A	19910404	(199116)*		
RW: AT BE CH DE DK ES FR GB IT NL SE					
W: CA FI HU JP NO SU US					
DD 298055	A5	19920206	(199227)		
EP 491858	A1	19920701	(199227)	EN	32
R: DE FR GB					
JP 05500512	W	19930204	(199310)		13
US 5336760	A	19940809	(199431)		14
WO 9104059	A3	19920220	(199510)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
DD 298055	A5		DD 1990-344026	19900914
EP 491858	A1		EP 1990-914864	19900914
			WO 1990-US5228	19900914
JP 05500512	W		JP 1990-513768	19900914
			WO 1990-US5228	19900914
US 5336760	A	Cont of	US 1989-407487	19890914
			WO 1990-US5228	19900914
			US 1992-838711	19920312
WO 9104059	A3		WO 1990-US5228	19900914

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 491858	A1	Based on	WO 9104059
JP 05500512	W	Based on	WO 9104059

PRIORITY APPLN. INFO: US 1989-407487 19890914
AN 1991-117337 [16] WPIDS
AB WO 9104059 A UPAB: 19930928
The following are claimed: (A) a method of prepg. a pharmaceutical compsn. of a specific target antibody component population in a pharmaceutically acceptable carrier from a physiological fluid in a chamber having at least one inlet and outlet comprising (a) bringing the physiological fluid into contact with a solid phase support bearing an **antigen component** selective for the target antibody component population, (b) removing the physiological fluid from the chamber, (c) washing the chamber with a wash fluid, (d) draining the wash fluid from the chamber, (e) supplying a pharmaceutically acceptable eluting soln. to the chamber and (f) and eluting the eluting soln. from the chamber.

USE/ADVANTAGE - The method can be used for selectively isolating a target antibody component population for the **treatment** of allergy or **autoimmune disease** . The method can also be used to isolate a factor VIII inhibitor complex for the **treatment** of haemophiliacs who have become refractory to the injection of factor VIII.
1/3

L5 ANSWER 23 OF 26 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1988-197939 [28] WPIDS
DOC. NO. NON-CPI: N1988-151226
DOC. NO. CPI: C1988-088371
TITLE: Immunoassay for detection of immune complexes - e.g. rheumatoid factor, and procedure for removing complexes from body fluids.
DERWENT CLASS: B04 S03
INVENTOR(S): ROPER, M D
PATENT ASSIGNEE(S): (BIOS-N) BIOSTAR MED PROD
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 4753893	A	19880628	(198828)*		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 4753893	A	US 1986-927609	19861106

PRIORITY APPLN. INFO: US 1985-740018 19850531; US 1985-763955 19850808; US 1986-927609 19861106
AN 1988-197939 [28] WPIDS
AB US 4753893 A UPAB: 19930923
An immunoassay of a body fluid specimen to determine the compsn. and/or concn. of immune complexes (I) present is claimed. The specimen and a combination (II), comprising immunologically nonspecific alkaline **treated** gammaglobulin (derived from animals not immunised against any antigenic determinant) and an additional fixative agent, namely anti-antibodies or rheumatoid factors, are introduced onto a receiving means (R) such that (II) adheres to the (R) and binds (I) which may be present in the specimen. The fixed (I) are then **treated** so as to deduce their compsn. and/or concn. and the antibody and **antigen components** of the specimen.

Also claimed is a method for removing (I) from body fluids using (II) fixed to a substrate so as to form a composite coating. In an embodiment (also claimed), a soln. contg. (I) and PBS is contacted with the fixed composite and adherence of (I) to the composite is promoted by incubation at a preselected temp. An article for effecting such removal is also provided.

USE/ADVANTAGE - The method may be used to detect rheumatoid factor (claimed), as well as for more general applicns., e.g., clinical detection, removal or concn. of (I). The process is simple and highly adaptable. The process is esp. suitable for removal of (I) from blood of patients with **autoimmune diseases**. The blood may then be returned to the patient.
0/12

L5 ANSWER 24 OF 26 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 1999:14570 CABA
DOCUMENT NUMBER: 990500079
TITLE: Granulomatous hepatitis caused by Q fever

Hepatitis granulomatosa por fiebre Q

AUTHOR: Gonzalez-Canudas, J. A.; Vega-R., B.; Nellen-Hummel, H.; Lisker-Halpert, A.; Laredo-Sanchez, F.

CORPORATE SOURCE: Departamento de Medicina Interna, Hospital de Especialidades, Centro Medico Nacional Siglo XXI, IMSS, Mexico, D.F., Mexico.

SOURCE: Gaceta Medica de Mexico, (1997) Vol. 133, No. 5, pp. 475-477. 14 ref.
ISSN: 0016-3813

DOCUMENT TYPE: Journal
LANGUAGE: Spanish
SUMMARY LANGUAGE: English

AB A case of fever and hepatic granuloma is described from Mexico which was originally diagnosed as viral hepatitis but was subsequently attributed to Q fever (caused by **Coxiella burnetii**). The patient was a 49-year-old insulin-dependent **diabetic** man who had not been in contact with domestic animals or non-pasteurized milk. Diagnosis was established by liver biopsy and confirmed serologically. Drug **therapy** with 500 mg tetracycline every 6 h for 10 days was successful. This case is unusual in that the patient did not show the typical clinical presentation of Q fever and the transmission of the disease was not established.

L5 ANSWER 25 OF 26 PHIN COPYRIGHT 2002 PJB

ACCESSION NUMBER: 2001:7980 PHIN
DOCUMENT NUMBER: C00705708
DATA ENTRY DATE: 10 Apr 2001
TITLE: PUBLICATIONS - New from Theta Reports - Vaccines 2001: The worlds market
SOURCE: Clinica-Online-plus (2001)
DOCUMENT TYPE: Newsletter
FILE SEGMENT: FULL

L5 ANSWER 26 OF 26 PHIN COPYRIGHT 2002 PJB

ACCESSION NUMBER: 1998:8113 PHIN
DOCUMENT NUMBER: S00577552
DATA ENTRY DATE: 22 Apr 1998
TITLE: Aquila acquires VacTex for $8 million
SOURCE: Scrip (1998) No. 2328 p13
DOCUMENT TYPE: Newsletter
FILE SEGMENT: FULL

(FILE 'MEDLINE' ENTERED AT 11:13:47 ON 07 JUN 2002)

```
L6          502 SEA FILE=MEDLINE ABB=ON  PLU=ON  "COXIELLA BURNETII"/CT
L7        34625 SEA FILE=MEDLINE ABB=ON  PLU=ON  "DIABETES MELLITUS,
                INSULIN-DEPENDENT"/CT
L8            1 SEA FILE=MEDLINE ABB=ON  PLU=ON  L6 AND L7


L6          502 SEA FILE=MEDLINE ABB=ON  PLU=ON  "COXIELLA BURNETII"/CT
L9        26071 SEA FILE=MEDLINE ABB=ON  PLU=ON  "AUTOIMMUNE DISEASES"/CT

L10           0 SEA FILE=MEDLINE ABB=ON  PLU=ON  L6 AND L9
```

Searcher : Shears 308-4994

L8 ANSWER 1 OF 1 MEDLINE
AN 1999221903 MEDLINE
TI Q fever vaccine on trial for type I diabetes.
AU Bonn D
SO MOLECULAR MEDICINE TODAY, (1999 Apr) 5 (4) 143.
Journal code: CMK; 9508560. ISSN: 1357-4310.

L7 34625 SEA FILE=MEDLINE ABB=ON PLU=ON "DIABETES MELLITUS, INSULIN-DEPENDENT"/CT
L9 26071 SEA FILE=MEDLINE ABB=ON PLU=ON "AUTOIMMUNE DISEASES"/CT

L11 1792 SEA FILE=MEDLINE ABB=ON PLU=ON "Q FEVER"/CT
L12 47841 SEA FILE=MEDLINE ABB=ON PLU=ON ANTIGENS/CT
L13 27 SEA FILE=MEDLINE ABB=ON PLU=ON L11 AND L12
L14 0 SEA FILE=MEDLINE ABB=ON PLU=ON L13 AND (L7 OR L9)

FILE 'CAPLUS' ENTERED AT 11:22:06 ON 07 JUN 2002
L15 24 SEA ABB=ON PLU=ON (QF OR Q FEVER)(S)ANTIGEN
L16 0 SEA ABB=ON PLU=ON L15 AND ((AUTOIMMUN? OR AUTO IMMUN?)(5A)(DISEAS? OR DISORDER) OR COXIELLOS? OR DIABET? OR IDDM)

FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO, VETU, VETB, CABA, AGRICOLA, PHIC, PHIN, TOXCENTER' ENTERED AT 11:23:31 ON 07 JUN 2002
L17 7 SEA ABB=ON PLU=ON L16
L18 4 DUP REM L17 (3 DUPLICATES REMOVED)
L19 3 SEA ABB=ON PLU=ON L18 NOT L5

L19 ANSWER 1 OF 3 MEDLINE
ACCESSION NUMBER: 96378956 MEDLINE
DOCUMENT NUMBER: 96378956 PubMed ID: 8784519
TITLE: Pore-forming activity of Coxiella burnetii outer membrane protein oligomer comprised of 29.5- and 31-kDa polypeptides. Inhibition of porin activity by monoclonal antibodies 4E8 and 4D6.
AUTHOR: Banerjee-Bhatnagar N; Bolt C R; Williams J C
CORPORATE SOURCE: Bacteriology Division, United States Army Medical Research Institute of Infectious Diseases Fort Detrick Frederick, Maryland 21702, USA.
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1996 Jul 23) 791 378-401.
Journal code: 5NM; 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961106
Last Updated on STN: 19970203
Entered Medline: 19961018

AB Envelopes of large-cell variant Coxiella burnetii, the agent of **Q fever**, were the starting material for purification of an outer membrane protein (OMP) oligomer with aggregate molecular mass of approximately 2 x 10(4) kDa. The oligomer was resistant to trypsin and dissociation by SDS at 100 degrees C. Reducing agents dissociated the oligomer into monomers of

29.5 and 31 kDa, which migrated as a doublet during SDS-polyacrylamide gel electrophoresis. Both monomers were reactive in an immunoblot assay with monoclonal antibodies (mAbs) 4E8 and 4D6, which were previously selected for their reactivity with purified and SDS-denatured 29.5 kDa protein. Proteoliposomes were functional in an equilibrium assay at pH 7 and a swelling assay at pH 7 and 4.5. The pores in proteoliposomes allowed the passage of arabinose, glucose, and sucrose, but restricted stachyose. Polyclonal antibodies to C. burnetii cells and the mAbs were able to bind C. burnetii at pH 7 and 4.5. The uptake of 14C-glucose at pH 4.5 was inhibited by polyclonal antibodies and mAbs after binding to cells at pH 7. The mAbs did not inhibit 14C-glucose uptake at pH 4.5 after binding to cells at pH 4.5. Although the mAbs bind C. burnetii porin epitopes before and after acid activation, the mAbs bound under acidic conditions were unable to inhibit porin function. The inhibition of porin channel function by mAbs confirms the role of porin as a permeability barrier for the subsequent active transport of glucose by C. burnetii. In another study, we showed that the 29.5 kDa OMP **antigen** induced active immunity against virulent challenge. This information, combined with the recent confirmation that porins are important **antigens** in the induction of specific protective immune responses against infection by gram-negative bacteria, suggests that humoral immunity directed against C. burnetii porins might play an important role in immunity against **Q fever** (human infection) and **coxiellosis** (animal infection), global enzootic diseases.

L19 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:95503 BIOSIS
DOCUMENT NUMBER: BA85:52275
TITLE: A CASE OF Q FEVER IN MORAVIA CZECHOSLOVAKIA.
AUTHOR(S): REHACEK J; PEJCOCH M; LISAK V; PRIVOROVA A; LOKAJ J; KAZAR J; PEJCOCHOVA J
CORPORATE SOURCE: VU SAV, MLYNSKA DOLINA 1, 817 03 BRATISLAVA.
SOURCE: CESK EPIDEMIOL MIKROBIOL IMUNOL, (1987) 36 (5), 280-286.
CODEN: CKEMAE. ISSN: 0009-0522.
FILE SEGMENT: BA; OLD
LANGUAGE: Slovak

AB The authors describe a case of the acute form of **Q fever** in a 49-year-old invalid living in a village. The source of infection was probably cattle of the local agricultural cooperative. In 25% of the cattle specific antibodies against the **antigen** of Coxiella burnetii phase II in the agglutination and complement fixation reaction were detected. In 12 of 35 examined members of the agricultural cooperative the skin test of late sensitivity with the **antigen** of Coxiella burnetii was positive. Five of these subjects had also a positive microagglutination reaction. The authors discuss the problem of latency of the focus of **coxiellosis** in agricultural institutions and the cause of sporadic manifestation of the disease.

L19 ANSWER 3 OF 3 VETU COPYRIGHT 2002 THOMSON DERWENT
ACCESSION NUMBER: 1989-63587 VETU M T
TITLE: Maintaining the Health of Animals with Healthy Feed. Survey of Biopreparations for Maintaining the Health of Sheep and Cattle.
(Ochranou zdravi zvirat ke zdrave potravine. Prehl'ad

biopreparatov pri ochrane zdravia oviec a hovadzieho dobytka)
AUTHOR: Kovac S; Lisak V
CORPORATE SOURCE: Bioveta
LOCATION: Nitra; Bratislava, Czech.
SOURCE: Veterinarstvi (39, No. 8, 340-41, 1989)
CODEN: VTERAT
AVAIL. OF DOC.: Bioveta Nitra, Czechoslovakia.
LANGUAGE: Slovakian
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
AN 1989-63587 VETU M T
AB The Authors survey several commercial biopreparations (enzootic abortion vaccine, **Q-fever** vaccine, listeriosis vaccine, E.coli vaccine, IBR, adenovirus and parainfluenza vaccine, and reovirus, adenovirus and parainfluenza vaccine) developed by Bioveta for the prophylactic vaccination and diagnosis of infectious disease in sheep and cattle. These specific vaccines and **antigens** have shown a broad spectrum of application in veterinary medicine based upon both literature data and the Authors' own experimental results. The value of these preparations resides in the reduction of stress factors that threaten animal health rather than on purely economic grounds.
ABEX Epobion has been developed as a vaccine against ovine enzootic abortion. It comprises a formaldehyde inactivated preparation of Chalmydia psittaci in isotonic saline, which is given s.c. to sheep over 3 mth old in early pregnancy. The preparation Epobion RVK is a Ch.psittaci antigen for the complement binding reaction. Bodibion MAR is a Coxiella burnetii antigen in phase II for a microagglutination reaction, which is used for determination of agglutinating antibodies in blood serum for **coxiellosis** in animals. Bodibion KFR is also a Coxiella burnetii antigen for complement binding reaction, while Bodibion is a vaccine against **coxiellosis** in sheep and cattle, which is formulated in isotonic saline-phosphate buffer for s.c. administration. Kombion is a combined vaccine against **coxiellosis** and enzootic abortion in sheep prepared from inactivated Cox. burnetii and Ch. psittaci in saline-phosphate buffers. Listakol is an inactivated oil vaccine for immunoprophylaxis of listeriosis in sheep of all age ranges. Bovibronchin is an inactivated oil vaccine against respiratory disease in cattle. The preparation contains adeno-1 and -3, parainfluenza virus 3 and reovirus 3 for i.m. injection into the neck and thigh regions. Polybronchin is a polyvalent vaccine for injection into cows in different stages of pregnancy where neonatal pneumonia is prevalent. The vaccine is formulated from IBR, parainfluenza 3, adeno-1 and reovirus 3 for raising colostral antibodies and in active immunization of calves. Plasmikol T is an inactivated vaccine against coli enteritis of calves in the early postnatal period for oral dosing. (Z46/CLW)

(FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO, VETU, VETB, CABA, AGRICOLA, PHIC, PHIN, TOXCENTER' ENTERED AT 11:27:36 ON 07 JUN 2002)

Author(s)

L20 542 S COWDEN W?/AU
L21 809 S LAFFERTY K?/AU
L22 37 S GAZDA L?/AU
L23 1 S L20 AND L21 AND L22
L24 1 S L20 AND (L21 OR L22)

Searcher : Shears 308-4994

L25 16 S L21 AND L22
L26 1371 S L20 OR L21 OR L22
L27 2 S L26 AND (L1 OR L15)
L28 17 S L23 OR L24 OR L25 OR L27
L29 7 DUP REM L28 (10 DUPLICATES REMOVED)

L29 ANSWER 1 OF 7 MEDLINE
ACCESSION NUMBER: 2002299734 IN-PROCESS
DOCUMENT NUMBER: 21980851 PubMed ID: 11983015
TITLE: Host systemic and local nitric oxide levels do not correlate with rejection of pig proislet xenografts in mice.
AUTHOR: Simeonovic Charmaine J; Cordery Damien V; Van Leeuwen Barbara; Popp Sarah K; Townsend Michelle J; Paule Michelle F; Wilson J Dennis; **Cowden William B**
CORPORATE SOURCE: Division of Molecular Medicine and Immunology and Cell Biology, The John Curtin School of Medical Research, Canberra, Australia..
Charmaine.Simeonovic@anu.edu.au
SOURCE: XENOTRANSPLANTATION, (2002 May) 9 (3) 169-82.
Journal code: 9438793. ISSN: 0908-665X.
PUB. COUNTRY: Denmark
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020604
Last Updated on STN: 20020604

AB The rejection of pig proislet xenografts in mice is a CD4 T cell-dependent process in which macrophages play an important role. To assess the potential for activated macrophages to act as effector cells in xenograft destruction, we have examined the relationship between proislet xenograft rejection, two principal markers of macrophage activation, transcription of inducible nitric oxide synthase (iNOS) and production of nitric oxide (NO), and their temporal relationship to intragraft cytokine gene expression. Xenograft rejection in CBA/H mice correlated with early induction of intragraft host iNOS mRNA and marked intragraft production of NO (reactive nitrogen intermediates, RNI). Intragraft mRNA expression for IFN-gamma, IL-1beta and TNF, cytokines associated with macrophage activation, was also found. These findings suggested that activated macrophages could be contributing to xenograft destruction via local NO-mediated toxicity at the graft site.To test the role of NO in this model: (1) **Q-fever antigen** (**QFA**) was administered to recipient mice in order to induce high systemic RNI levels and (2) in another experiment, pig proislets were transplanted into iNOS-/- mice. Treatment with **QFA** correlated with prolonged xenograft survival at 7 days post-transplant. Splenocytes from **QFA**-treated, but not control mice at 7 and 22 days post-transplant, exhibited inhibition of secondary xenogeneic mouse antiporcine mixed lymphocyte reaction (MLR) that was reversed by culture with the NOS inhibitor N-methylarginine (NMA). Despite continued elevated NO production, xenograft protection was temporary with complete rejection by day 22. Evidence that locally produced NO was not contributing to rejection was seen when pig proislets transplanted into iNOS-/- mice were rejected with normal kinetics; in these animals intragraft NO production was not detected (despite porcine iNOS gene expression). Failure of activated macrophages to achieve indefinite xenograft

survival suggests that other factors are also required. Macrophage potential to effect either destructive or protective roles after pig proislet xenotransplantation suggests that such functions may depend on the site and magnitude of macrophage activation. Together these findings clearly demonstrate that high NO levels in the periphery are not damaging to xenogeneic islet tissue, neither host nor donor NO production is essential for islet xenograft rejection and consequently elevated plasma RNI levels do not represent a direct marker for rejection.

L29 ANSWER 2 OF 7 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 1997-470646 [43] WPIDS
DOC. NO. CPI: C1997-149552
TITLE: Prevention or treatment of auto-immune disease using **Coxiella** or derived antigens - especially for insulin-dependent diabetes, also to improve survival of transplanted islet cells.
DERWENT CLASS: B04 D16
INVENTOR(S): **COWDEN, W B; GAZDA, L S; LAFFERTY, K J**
PATENT ASSIGNEE(S): (AUSU) UNIV AUSTRALIAN NAT; (COWD-I) COWDEN W B; (GAZD-I) GAZDA L S; (LAFF-I) LAFFERTY K J
COUNTRY COUNT: 78
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9733614	A1	19970918	(199743)*	EN	34
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU					
AU 9719171	A	19971001	(199805)		
ZA 9702232	A	19980128	(199810)		35
EP 886528	A1	19981230	(199905)	EN	
R: AL AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC NL PT RO SE SI					
JP 2000506172	W	20000523	(200033)		33
AU 720327	B	20000525	(200034)		
NZ 331536	A	20000728	(200043)		
NZ 503143	A	20010629	(200140)		
US 2001051162	A1	20011213	(200204)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9733614	A1	WO 1997-AU161	19970314
AU 9719171	A	AU 1997-19171	19970314
ZA 9702232	A	ZA 1997-2232	19970314
EP 886528	A1	EP 1997-906937	19970314
		WO 1997-AU161	19970314
JP 2000506172	W	JP 1997-532124	19970314
		WO 1997-AU161	19970314
AU 720327	B	AU 1997-19171	19970314
NZ 331536	A	NZ 1997-331536	19970314

			WO 1997-AU161	19970314
NZ 503143	A		NZ 1997-503143	19970314
US 2001051162	A1	Cont of	WO 1997-AU161	19970314
		Cont of	US 1999-142597	19990305
			US 2001-887773	20010621

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9719171	A	Based on	WO 9733614
EP 886528	A1	Based on	WO 9733614
JP 2000506172	W	Based on	WO 9733614
AU 720327	B	Previous Publ.	AU 9719171
		Based on	WO 9733614
NZ 331536	A	Based on	WO 9733614
NZ 503143	A	Div in	NZ 512067

PRIORITY APPLN. INFO: AU 1996-8703 19960314
AN 1997-470646 [43] WPIDS
AB WO 9733614 A UPAB: 19971030
The effect of an autoimmune disease in a mammal is prevented, inhibited, delayed or otherwise alleviated by administration of a **Coxiella** species, or one of its antigens (or analogues or homologues) (I). Also claimed are: (i) a method for prolonging the survival of transplanted islet tissue comprising administration of (I); and (ii) a composition comprising (I).

USE - The method is used in humans or other animals, specifically to treat or prevent insulin-dependent diabetes mellitus (but may also be effective in pernicious anaemia, chronic hepatitis, ulcerative colitis, primary biliary cirrhosis, multiple sclerosis and systemic lupus erythematosus) or a disease that affects survival of transplanted pancreatic islet tissue.

ADVANTAGE - (I) is sufficiently safe for general use (contrast complete Freund's adjuvant (FCA) or bacillus Calmette-Guerin) and is more effective at diverting the immune response away from destructive autoimmunity.

Dwg.1/2

L29 ANSWER 3 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 97361792 MEDLINE
DOCUMENT NUMBER: 97361792 PubMed ID: 9218753
TITLE: Diabetes results from a late change in the autoimmune response of NOD mice.
AUTHOR: **Gazda L S**; Charlton B; **Lafferty K J**
CORPORATE SOURCE: Division of Molecular Medicine, John Curtin School of Medical Research, Australian National University, Canberra.
CONTRACT NUMBER: DK46621 (NIDDK)
SOURCE: JOURNAL OF AUTOIMMUNITY, (1997 Jun) 10 (3) 261-70. Journal code: ADL; 8812164. ISSN: 0896-8411.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970902
Last Updated on STN: 19970902

Entered Medline: 19970819

AB IDDM in the NOD mouse is the result of a chronic autoimmune process. NOD mice are shown to express benign autoimmunity that converts to a state of malignant autoimmunity and the development of IDDM. Young disease-prone NOD mice are in a state of benign autoimmunity that is correlated with a non-destructive response to islet tissue and the preservation of insulin-containing beta-cells. A proportion of mice with benign autoimmunity convert to having malignant autoimmunity. Clinical diabetes is diagnosed approximately 3 weeks from the development of malignant autoimmunity which is correlated with a destructive response to grafted islet tissue and extensive beta-cell destruction. We conclude that the development of clinical disease is correlated with a change in the state of autoimmunity, that is, from benign to malignant autoimmunity.

L29 ANSWER 4 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97232285 MEDLINE
DOCUMENT NUMBER: 97232285 PubMed ID: 9077560
TITLE: Tolerance: a case of self/not-self discrimination maintained by clonal deletion?.
AUTHOR: **Lafferty K J; Gazda L S**
CORPORATE SOURCE: Division of Molecular Medicine, John Curtin School of Medical Research, Australian National University, Canberra.
CONTRACT NUMBER: DK-94-017 (NIDDK)
DK46621 (NIDDK)
SOURCE: HUMAN IMMUNOLOGY, (1997 Feb) 52 (2) 119-26. Ref: 45
Journal code: G9W; 8010936. ISSN: 0198-8859.
PUB. COUNTRY: United States
Historical
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970709
Last Updated on STN: 19970709
Entered Medline: 19970623

AB The clonal selection theory of immune reactivity is based on a metaphor of self/not-self discrimination and considers self-tolerance to result from clonal deletion. There is evidence that a deletional mechanism is responsible for negative selection of self MHC-reactive T-cells in the thymus. Bretscher/Cohn theory builds on this concept and provides a model which allows self/not-self discrimination to occur at any time throughout the life of the individual. However, modern concepts of antigen presentation in which MHC-peptide co-presentation is the unit recognised by the T-cell receptor have abandoned the Bretscher/Cohn requirement for associative recognition of antigen. For this reason, such models of APC function cannot use Bretscher/Cohn theory to explain self/not-self discrimination. Matzinger's 'danger' metaphor for the immune system provides a theoretical way forward by moving the emphasis away from an immune system based on self/not-self discrimination. These theoretical developments lead to a novel approach to the control of autoimmunity that is based on the strengthening of immune regulation by the use of adjuvant therapy.

L29 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:372948 BIOSIS
DOCUMENT NUMBER: PREV199799672151
TITLE: BCG vaccine modulates autoimmune process in newly diagnosed IDDM patients (preliminary report.
AUTHOR(S): Vazeou, A.; **Lafferty, K.**; Pergantou, E.; Spanos, E.; **Gazda, L.**; Bartsocas, C.
CORPORATE SOURCE: Dep. Pediatrics, Faculty Nursing, Athens Univ., Athens Greece
SOURCE: Hormone Research (Basel), (1997) Vol. 48, No. SUPPL. 2, pp. 7.
Meeting Info.: 5th Joint Meeting of the European Society for Paediatric Endocrinology and the Lawson Wilkins Society for Pediatric Endocrinology, in Collaboration with the Australian Paediatric Endocrine Group, the Japanese Society for Pediatric Endocrinology and the Latin American Society for Paediatric Endocrinology Stockholm, Sweden June 22-26, 1997
ISSN: 0301-0163.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L29 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
ACCESSION NUMBER: 1996:696528 HCAPLUS
DOCUMENT NUMBER: 126:6272
TITLE: Regulation of autoimmune diabetes: Characteristics of non-islet-antigen specific therapies
AUTHOR(S): **Gazda, Lawrence S.**; Baxter, Alan G.; **Lafferty, Kevin J.**
CORPORATE SOURCE: John Curtin School Medical Research, National University, Canberra, Australia
SOURCE: Immunol. Cell Biol. (1996), 74(5), 401-407
CODEN: ICBIEZ; ISSN: 0818-9641
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Non-islet-antigen specific treatments have been shown to alter the natural history of insulin dependent diabetes in both the non-obese diabetic (NOD) mouse and in recently diagnosed patients. However, concerns have been raised regarding the possibility that non-islet-antigen specific therapy may trade cell mediated autoimmunity for antibody dependent autoimmunity. Female NOD mice at approx. 70 days of age were treated with the non-islet-antigen specific agents complete Freund's adjuvant (CFA) and Bacillus Calmette-Guerin (BCG) and assayed for the development of antibody mediated autoimmunity at 300 days of age. Autoantibodies to red cells were not detected in any of the BCG (n = 19) or CFA (n = 15) treated animals, while 2 of 13 age-matched NOD animals had autoantibodies to red cells, shown by a pos. direct Coomb's test. Anti-nuclear autoantibodies and complement deposition in the renal glomeruli were not significantly increased in the treated animals as compared to age-matched non-diabetic mice. The relative effectiveness of CFA and BCG treatment was examd. in terms of the ability of these agents to preserve insulin contg. islets. Complete Freund's adjuvant treatment was found to be more effective in preserving insulin contg. islets when compared to BCG treatment.

This study demonstrates that it is possible to inhibit the development of autoimmune diabetes without increasing the probability that treated animals will develop antibody dependent autoimmunity.

L29 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
ACCESSION NUMBER: 1996:151550 HCAPLUS
DOCUMENT NUMBER: 124:229092
TITLE: Autoimmune diabetes: caught in the causality trap?
AUTHOR(S): **Gazda, Lawrence S**; Gilchrist, Kirsty A; **Lafferty, Kevin J**
CORPORATE SOURCE: John Curtin School of Medical Research, Australian National Unversity, Canberra, 2601, Australia
SOURCE: Immunol. Cell Biol. (1995), Volume Date 1995, 73(6), 549-51
CODEN: ICBIEZ; ISSN: 0818-9641
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 14 refs. The lack of concordance between genotype and clin. diabetes has prompted a search for the infectious agent that ppts. this autoimmune disease. However, this approach may be misleading. It assumes that the disease-prone individuals that do not develop diabetes do not have autoimmunity. In the non-obese diabetic (NOD) mouse, the genotype is a primary determinant of autoimmunity. Not all animals of the disease-prone genotype develop clin. disease; however, all have autoimmunity. This is expressed as a destructive or non-destructive process. Multiple pathways are open to the immune system and whether or not the immune response is destructive and leads to the development of clin. disease, appears to be a random process. If this is the case, the most important questions relating to autoimmune disease are not those concerning the 'causative' agents. Instead the authors should be asking what are the differences between pathways open to the immune system and what factors affect the probability that one or another pathway is finally selected.

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FILE 'HOME' ENTERED AT 11:34:14 ON 07 JUN 2002